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VASCULAR AND NEUROLOGIC LESIONS IN SURVIVORS OF SHIPWRECK*

I. Immersion-Foot Syndrome Following Exposure to Cold

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As a result of enemy submarine activity, many survivors of torpedoed vessels have been exposed to unusual physiologic conditions. These include cold air and water and, when rescue is long deferred, prolonged dependency of the legs, malnutrition and vitamin deficiency. Many of these men have required hospitalization in naval and marine hospitals. One of the commonest incapacitating lesions is painful swelling of the feet and lower legs. This condition has been called "immersion foot." It has been my privilege to study it in Scotland and along the western Atlantic seaboard from Nova Scotia to Virginia. A surprising feature is that painful swelling of the lower extremities may take place both after exposure to the cold of the North Atlantic winter and the warmth of the Gulf Stream in southern latitudes in April and May.

The purpose of this paper is to point out that in the true immersion-foot syndrome, which occurs after relatively brief exposure of the lower extremities to cold water, there is actual thermal injury to the skin and subcutaneous tissue; whereas in the survivors of prolonged exposure in southern waters this feature is not present, but the painful swelling of the dependent parts is caused by a systemic disturbance.

CLINICAL OBSERVATIONS

Immersion foot, caused by prolonged chilling just short of actual freezing, has not heretofore received adequate recognition, although it must have occurred after torpedoings in the last war and after shipwreck in northern waters since time immemorial. An excellent clinical picture has recently been given by Webster, Woolhouse and Johnston,[1] the Canadian naval medical officers who have treated by far the largest number of cases According to them:

> At the time of removal of the patients from the open boats or rafts, the feet were cold, swollen and waxy white in color, with scattered cyanotic areas. The patients complained at that time that their feet felt heavy, "woody" and numb, and the feet were anaesthetic to pain, touch and temperature.
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> Shortly after removal of the feet from this traumatizing environment, the swelling increased rapidly as the feet became red, hyperaemic and hot without sweating, and the pulse in the vessels of the feet was full and bounding. In those more severely damaged areas — usually the toes, the distal part of the dorsum of the foot and the ball of the foot — the parts remained oedematous and hot, and assumed a livid, cadaveric appearance. A number of these latter areas later produced blebs, some of which were filled with straw-colored fluid and others with extravasated blood; and areas of ecchymosis commonly appeared over the medial aspect of the first metatarsophalangeal joint and on the medial aspect of the longitudinal tarsal arch. Many of these feet presented the appearance of incipient massive gangrene. The rapid swelling and increasing redness, accompanied by the extreme rise in temperature of the part, presented the picture of an intense vasodilatation, accompanied by definite damage to the vascular wall with transudation, as manifested by bleb formation and extravasation of blood.
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> The anaesthesia followed a variety of patterns. In the mildest cases these areas extended around the margins of the foot and over its plantar aspect, being of greater extent along the lateral border, where the mass-to-surface ratio is smallest. In the more severe cases these areas extended over the entire dorsum of the foot, and in some cases over the lower two thirds of the leg. Patches of anaesthesia over the knees in the prepatellar and infrapatellar regions were observed in two cases where there had been prolonged kneeling in the lifeboats.

Attention was first focused on this syndrome by the Medical Corps of the Royal Navy after the loss of a ship off the northern Norwegian coast in the late spring of 1941. The ocean temperature was around 40°F. A number of the crew were adrift on Carley rafts for several days. These rafts are large, doughnut-shaped floats with an open lattice in the center. The occupants are therefore forced to sit on the floating outer hull and keep their feet immersed in the cold water that washes in

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and out of the bottom. After these survivors were rescued by trawlers (which carry no medical officers), the mistake was made of rapidly warming their numb and swollen feet before the galley stove. Three months later, when I visited the naval hospital at which they had been treated, the last of these patients was being discharged. From the description given me by Surgeon Rear Admiral Sir William Wheeler and from the recently published account by Ungley,[2] the vascular and neurologic changes in the immersed extremities were identical with those of the Halifax survivors that are described below, except for their greater severity. Owing to the unfortunate rapid warming before the galley stove, a number of these men developed gangrene, which necessitated amputation. The others suffered from trophic disturbances of the skin and neuritic pain, which prevented their return to active duty for a very protracted period. The severity of the process and the prolonged period of convalescence illustrate the need of efficient first-aid and subsequent hospital treatment.

During the past winter over 100 similar cases have been treated in Halifax. Through the kindness of Surgeon Commander D. W. Johnstone, the senior medical officer of the Canadian Navy there, I was given every facility to examine 32 survivors of three vessels torpedoed in the North Atlantic early in March, 1942. These patients were under the charge of Surgeon Lieutenant Commander D. R. Webster, Surgeon Lieutenant F. M. Woolhouse and Surgeon Lieutenant J. L. Johnston, who were extremely co-operative and helpful. A description of their cases has just been published by them.[1] The reason for presenting this brief résumé of their work is twofold: first, to add observations on 15 other cases seen at the Halifax Infirmary, with the senior surgeon, Dr. Alan Currie, at the Columbia Medical Center, with Dr. David Bull, and at the United States Naval Hospital in Chelsea; secondly, to contrast these cases of immersion in cold water with a series of superficially similar, but fundamentally quite different, results of more prolonged immersion in warm water seen at the United States Marine Hospital in Norfolk during May, 1942. The latter group will be taken up in the second part of this paper.

The 32 patients seen in Halifax were exposed to air and water close to freezing temperature in three lifeboats for periods ranging from twenty-seven hours to four days. In all cases it had been impossible to keep the boats bailed dry, so that water was sloshing about over the floor boards from a depth of a few inches up to the height of the thwarts. The men's legs were therefore constantly wet below the knees and actually immersed most of the time from the ankles down in cold water. The reaction of the lower extremities to cold and wet was greatly aggravated by the fact that they were continually dependent and that, on account of crowding, there was no opportunity to exercise or elevate the legs (see below, section on "General Responses of Tissues to Cold"). Whether the survivors wore shoes or not made little difference, as the wet feet became so swollen that any boot soon became tight and constricting. The feet under these conditions rapidly became numb, painless and cadaveric in color. A few of the men tried at first to keep out of the water, but soon gave up. Only in the rare cases where men had on loosely fitting rubber hip boots and managed to keep their feet relatively dry, or where they were able to wrap them in a blanket and keep them clear of the water for the better part of the time, were they able entirely to avoid this condition.*

On being picked up by rescuing vessels, those who suffered from immersion foot observed that it felt as though they were "walking on air" or "on blocks of wood." The more severely afflicted men could not maintain their balance or walk, and had to be supported or carried. On arriving in warm quarters below decks, their feet were so swollen that if boots had been kept on they usually had to be cut off. The extreme swelling and gray, cyanotic hue of the severe cases made it look as though many might develop gangrene of the feet. Fortunately, the mistake made in the first-aid treatment of the British survivors reported above was not repeated here. None of these men had their numb feet soaked in hot water or warmed before a galley stove. Some had them immersed in cold water for a brief period, others rubbed with alcohol (even this was a mistake; see below, section on "Treatment"). They were then put in bunks and given hot drinks. In the milder cases the feet began to tingle and burn as soon as they warmed up, but those more severely chilled remained numb for much longer periods.

On reaching Halifax it was observed that the feet, which at the time of rescue had been cadaveric in color, cold and numb, were pink, hot and painful, as in erythromelalgia. The edematous skin was dotted with petechial hemorrhages and, in the severer cases, with large blisters that resembled those of a superficial burn. Sensation,

*Since this article was written I have had the opportunity of examining 8 members of the crew on a torpedoed merchantman after three days adrift in cold water. Three of these men had been protected by a new type of rubber lifesuit and suffered no thermal injuries, whereas the remainder, who wore ordinary clothes, all developed immersion foot of moderate severity.

which at first was absent above the ankle, usually descended to the midfoot within a period of twenty-four hours, although the toes often remained anesthetic for a period of several weeks (Fig. 1). With the partial return of sensation, tion secondary to epidermophytosis eventually necessitated their amputation. Eight patients showed edema, blebs, petechiae and superficial patches of gangrene from their severe chilling (Cases 2, 3, 4, 6, 7, 8, 18 and 19). Thirteen men (including all

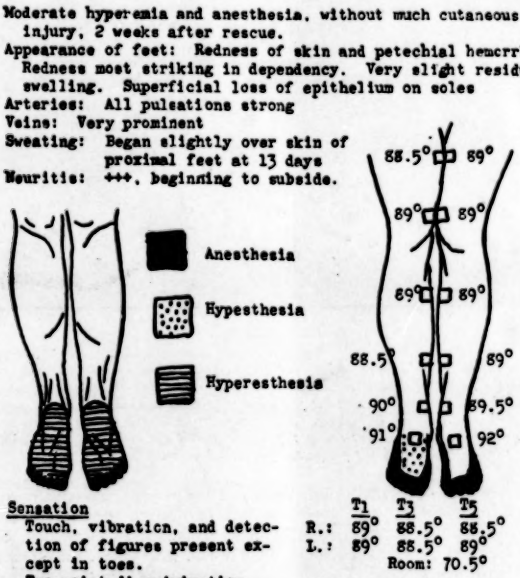
Moderate hyperemia and anesthesia, without much cutaneous injury, 2 weeks after rescue.
Appearance of feet: Redness of skin and petechial hemorrhages
Redness most striking in dependency. Very slight residual swelling. Superficial loss of epithelium on soles
Arteries: All pulsations strong
Veins: Very prominent
Sweating: Began slightly over skin of proximal feet at 13 days
Neuritis: +++, beginning to subside.
Anesthesia
Hypesthesia
Hyperesthesia
88.5° 89°
89° 89°
89° 89°
88.5° 89°
90° 89.5°
91° 92°
Sensation
Touch, vibration, and detection of figures present except in toes.
Two-point discrimination slightly reduced over dorsum of feet, absent in toes
T1 T3 T5
R.: 89° 88.5° 88.5°
L.: 89° 88.5° 89°
Room: 70.5°

FIGURE 1. *Case 10.*

burning, tingling pain became intense and, together with the risk of the blebs' rupturing and becoming infected, presented the major therapeutic problem. These clinical findings seen in the Halifax cases are summarized in Table 1 and illustrated in Figures 2 and 3.

Examination of Table 1 shows that of the 19 acute cases of true immersion foot examined in detail, only 2 developed areas of gangrene of sufficient depth to require surgery. In Case 19 bilateral major amputations were necessary, owing to the cellulitis that followed the prolonged severe exposure and was already present when the patient reached the hospital. The infection entered through the blisters that always develop after long immersion in cold water. In Case 1 the loss of tissue involved several of the small toes, and late infec-

the above and also Cases 5, 9, 10, 13 and 14) had profound circulatory disturbances with intense hyperemia. The intensity of the hyperemia seemed to correspond directly to the degree of sensory impairment. The inability of the hyperemic feet to sweat was also a very constant observation, and indicates that injury of the sympathetic fibers corresponded to that of the pain fibers. As these two sets of axones closely resemble each other in their diameter, degree of myelinization and rate of conduction, this is not surprising. It was also evident that patients who showed the most definite signs of nerve and tissue damage were generally the ones that developed the severest degrees of neuritic pain. Pain was a distressing symptom in 11 patients (Cases 2, 3, 4, 6, 7, 9, 10, 11, 12, 13 and 18), particularly in the first two weeks. At the

end of this period the active inflammatory process was usually subsiding, and the skin of the sole and toes desquamating in large sheets. In cases with inadequate early treatment, both swelling and pain tended to persist and become a cause of prolonged disability (up to six months).

FIGURE 2. *Case 1.*

The photographs were taken on the twelfth day after rescue. The man had been in a lifeboat without shoes for four days in the North Atlantic in March. The temperature of the sea water was recorded at 34° F., and his feet had been wet most of the time. On arrival in Halifax the hyperemia and vascular engorgement of the lower legs were intense; his feet were greatly swollen, blistered and anesthetic; his toes were blue-black and gangrenous looking. After treatment by cooling for twelve days in ice-packs, the swelling was greatly reduced, the blebs had nearly absorbed, and the condition of the toes was distinctly improved. Nerve recovery had not progressed far enough to cause pain.

A number of late complications have been observed in consultation with Drs. A. W. Stearns and R. S. Schwab at the United States Naval Hospital in Chelsea, and with Dr. D. C. Bull at the Columbia-Presbyterian Medical Center in New York City. These include atrophy of the intrinsic muscles in the feet (2 cases), a chronic sense of bursting fullness in the lower legs in patients who had varicose veins (3 cases) and various manifestations of psychoneurosis (8 cases). The latter are outside the scope of this paper.

General Responses of Tissues to Cold

Before discussing the important subject of treatment, it will serve to orient one's concepts in that regard to review what is already known about the fundamental responses of the tissues to cold and the pathologic changes that take place in them after severe chilling and ischemia. On reviewing the literature, one is struck by the fact that there is no fundamental clinical or pathological difference in the varied syndromes that follow exposure to cold. There is furthermore a striking similarity to injuries due to burns. Modern accounts begin with Baron Larrey's[3] experiences while serving as surgeon-general of Napoleon's army in the winter retreat from Moscow. In the Crimean War[4] it was observed that the soldiers suffered most in temperatures just above freezing in the wet mud and snow of the trenches, particularly when unable to move about. Even then it was recognized that fatigue, stagnation of circulation, constriction of the legs and scurvy were important accessory factors. Greene,[5] who as medical officer of two Himalayan expeditions had an unusually wide experience, has written a most timely account of the injuries that follow various forms of exposure to cold. He describes the trench-foot syndrome of the last war, which is essentially identical with that of immersion foot. Numbness gradually develops in cold, wet, dependent extremities, and is followed by swelling and blistering. When the blisters rupture, streptococcal infection may bring about widespread gangrene. This may occur in lifeboats as well as in the trenches, provided the exposure is prolonged enough for the blisters to break and become infected, as in Case 19. Pain in trench foot also is a distressing symptom. "Shelter foot," first described in 1940 by Knight,[6] occurs in persons who spend nights in cold air-raid shelters without moving or elevating their legs. The condition is aggravated by constriction, as when the back of the calf has been compressed by the bar of a deck chair. These feet become red, shiny and painful. Increased capillary permeability from malnutrition (lowered serum protein and vitamin

deficiency) are important accessory factors. Greene makes a brief reference to immersion foot, but states that he has never seen a case of it. The first comprehensive reports on this syndrome have been published recently.[1, 2] In addition, previous clinical and laboratory investigations are of value in understanding the underlying features of the condition.

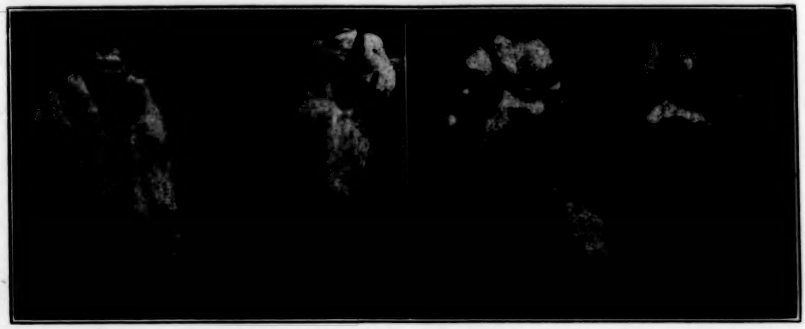

FIGURE 3. *Case 2.*

This seaman was in the same lifeboat as the man whose feet are shown in Figure 2. He also had no shoes, but his local injury from cold was much less pronounced. He was able to walk, and on admission to the hospital his feet were only slightly swollen, with hypesthesia of toes and intense hyperemia. Pain was severe, but after ten days' treatment in ice-packs, circulation had returned to normal and the pain was no longer present so long as his feet were exposed to the cool air of the ward. The patches of gangrene shown in the photographs, taken on the twelfth day, were only skin deep.

The effects of cold with special reference to frostbite were studied by Rischpler[7] in 1900. He froze animal tissues by the application of ice and salt, and investigated the results microscopically at various intervals. Under these circumstances actual degeneration was found in the cells of the blood-vessel walls, with swelling of the connective tissue and vacuolation of the smooth-muscle fibers, but there was no sign of thrombosis. During the last war Lake[8] found that the critical temperature where all metabolic processes are inhibited is -6°C. (21°F.). He claims that at this temperature blood freezes with destruction of cells, liberating thrombokinase, and that on thawing thrombosis occurs. More recent studies by Lewis[9] show that this is not necessarily true. Although the freezing point of blood is -0.53°C., and that of the tissues close to this level, Lewis found that skin when cold rarely freezes at its true freezing point. It exhibits the property of supercooling and may not freeze until the temperature falls to much lower levels, -20°C. (-4°F.). As the freezing point of sea water itself is -1.9°C., it is obvious that, in immersion foot, actual freezing does not take place.

In investigating trench foot in the last war, Smith, Ritchie and Dawson[10] carried out an admirable set of animal experiments under conditions that closely resemble those resulting in immersion foot. They subjected rabbits in a cold chamber to standing on wet mud. When this was continued for three days, especially after removing the fur from the paws, marked swelling of the feet resulted. Histologic examination showed changes in the blood-vessel walls, with an outpouring of fluid containing both cells and fibrin. Occasionally the nerve trunks showed diffuse swelling of the axis cylinders, but without evidence of degenerating fibers in preparations stained by the Marchi method. The investigations thus proved that simple prolonged exposure to cold can produce subacute inflammation of the skin and subcutaneous tissues of the feet. These changes could be exaggerated by moisture, by applying constricting bands to the upper legs and by rapidly warming the chilled extremities before the animals were sacrificed.

The most illuminating discussion of the pathologic physiology of tissues exposed to cold is contained in a series of three lectures delivered by Sir Thomas Lewis[9, 11] in 1941. On exposure to cold the blood vessels are constricted both by vasoconstrictor impulses from the hypothalamus and by direct contraction of the smooth-muscle

TABLE 1. *Summary of Findings in Cases of Immersion-Foot Syndrome (Northern Survivors).*

Case No.	Tissue Damage on Arrival at Halifax	Pain			Sensory Loss	Sweating	Hyperemia	Treatment	
		First Sensation	Severity	Duration				Type	Duration
1	Swelling and blebs ++; gangrene of several toes.	Still absent	?	?	Distal half of feet	Absent below ankles	+++	Continuous ice packs Later, amputation of several toes	12 days
2	Swelling and blebs +; patches of cutaneous gangrene.	4 days	++	Subsiding	Toes	Normal	+++ (10 days)	Continuous ice packs Exposure to air	10 days 2 days
3	Swelling and blebs ++; small petechiae.	5 days	+++	Beginning to subside	Distal third of feet	Absent below ankles	+++	Intermittent ice, air and electric fan	12 days
4	Swelling and blebs +; petechiae.	—	++	Beginning to subside	Toes	Absent lower feet and toes	+++	Intermittent ice, air and electric fan	12 days
5	Slight	—	+	Beginning to subside	Toes	Absent lower feet and toes	+++	Exposure to air	12 days
6	Swelling and blebs +	2 days	++	Still present	Toes (hypesthesia)	Absent lower feet and toes	+++ → +	Continuous ice packs	12 days
7	Swelling and blebs +	1 day	++	Still present	Toes (hypesthesia)	Normal	0	Exposure to air	12 days
8	Swelling and blebs +	1 day	++ → +	Subsiding	Almost gone	Normal	+	Exposure to air	12 days
9	Swelling +	3 days	++	Still present	Almost gone	Absent lower feet and toes	+++ → +	Continuous ice packs Exposure to air	13 days 1 day
10	Swelling +	10 days	+++	Increasing	Distal third of feet	Normal	+++	Exposure to air Intermittent ice packs	12 days 2 days
11	Slight	1 day	++	Subsiding	Toes (hypesthesia)	Normal	0	Exposure to air	12 days
12	Slight	12 hours	+ → ++	Still present	Toes (hypesthesia)	Increased	0	Exposure to air	12 days
13	Slight	1 day	+ → ++	Increasing	Toes (hypesthesia)	Normal	+	Continuous ice packs Exposure and elevation	3 days 10 days
14	Slight	3 days	?	?	Distal third of feet	Normal	+++	Continuous ice packs (right), exposure to air (left)	1 day
15	Slight	6 hours	+	?	Feet (patchy areas of hypesthesia)	Normal	0	Exposure to air	1 day
16	Slight	Never lost	Slight	?	0	Normal	0	Exposure to air	1 day
17	Slight	Never lost	Slight	?	0	Normal	0	Exposure to air	1 day
18	Swelling and blebs +; petechiae.	12 hours	+++	Still present	Distal half of feet	Absent below ankles	+++	Intermittent ice packs Exposure to air	14 days 1 day
19*	Cellulitis and septic gangrene of feet	—	—	—	—	—	—	Early amputations	

*Patient seen four months after rescue; reaction to immersion altered by infectious gangrene, which came on during thirteen days' exposure.

cells in their walls. But when the temperature of the skin drops near to freezing, there is an intermittent increase in blood flow, which serves to maintain the viability of the skin. This phenomenon is carried out by a special mechanism of arteriovenous anastomoses (Grant and Bland[12]) and is mediated by local axone reflexes. How long this mechanism can maintain its efficiency in the cold is not known, and it seems probable that the circulation must be reduced to a low point. Lewis observed that immersion of the normal hand or foot in water at 5°C. (41°F.) for as short a period as two hours resulted in definite swelling. This swelling, which may amount to as much as a 15 per cent increase in volume, is not the simple result of imbibition of water by the skin, since it happens in a hand protected by grease or covered by a rubber glove. If swelling of this degree can occur after a few hours, it is obvious that large amounts of fluid must be lost from the circulation on prolonged general exposure. The reduction in the circulating blood volume is the cause of the somnolence and death that overtake so many seamen in lifeboats during severe winter weather. Lewis considers this response to cold to be caused by an "H," histamine-like substance released from the injured cells and acting on the blood vessels.

When the chilled extremity is removed from ice water, the invariable response is a state of hyperemia. The local reddening, vasodilatation and extravasation of fluid from the injured vascular bed are all part of Lewis's classic "triple response." The wheal which first forms may be replaced by a blister. These changes together constitute a simple acute inflammation. Sections of human skin examined by Lewis twenty-four hours after chilling displayed edema of both epidermis and dermis, and a perivascular infiltration of the superficial layers of the dermis with lymphocytes, extravasated red blood cells and some polymorphonuclear leukocytes. Observations now completed in Halifax on many cases of immersion foot show that this inflammatory hyperemia reaches its height on about the third day after the period of exposure is ended and gradually disappears in the course of a fortnight, much as occurs in any other sterile inflammatory reaction.

The changes that occur in the nerve trunks and the terminal superficial axones to the skin are a second point of particular interest. These patients at first have complete cutaneous anesthesia below the ankles. In the mild cases this clears rapidly on warming, but in the severe ones there is still anesthesia of the toes at the end of a fortnight and reduced sensation over the remainder of the foot. The speed at which sensory recovery may take place over the dorsum of the foot is too great for nerve regeneration and is evidence that the majority of nerve fibers never actually degenerate. This fits in with Smith, Ritchie and Dawson's[10] observations in rabbits' feet after seventy-two hours' exposure to cold. They found definite swelling of the axones, but no evidence of their degeneration. However, in some of the patients of this series (Cases 1, 3 and 10) the toes were still anesthetic at the end of a fortnight and there was little sweating below the ankle. This suggests actual degeneration of the sensory and sympathetic axones to the more exposed distal portions of the feet, which may require a period of several months for complete regeneration and restoration of normal function. In Ungley's[2] recent report on the Scottish cases, Wallerian degeneration of some of the cutaneous nerves was observed by Blackwood, who examined the biopsies. It is a well-known fact that a partially injured nerve is likely to produce a painful neuritis, and this has been the universal experience in the severe cases of immersion foot. Accurate objective determinations of the recovery of nerves can be made by testing the return of sweating by the method recently described by Minor[13] and by Guttmann[14] at Oxford, and the changes in electrical skin resistance (Richter and Woodruff[15]). These methods will be utilized when suitable patients are available in Boston.

The fact that patients continue to suffer after their circulation has returned to normal suggests that the pain is neuritic in origin. This is the most logical explanation in the late stages, but during the acute phase of the reaction this may not be the entire reason. In this connection it is of interest to quote the impression of Webster and his colleagues[1] that these men are comfortable when their foot temperature is kept below 80°F., but that as soon as the ice packs are left off and the cutaneous temperature of the toes is allowed to exceed 90°F. they again experience pain. This fits in with the observations made by Freeman[16] on legs with threatened gangrene. By plethysmographic studies of blood flow in sympathectomized extremities, he has demonstrated that a rise with local heating is caused solely by the increased metabolism of the tissue cells and their need for more blood and oxygen. Local cellular metabolism, like other chemical reactions, should double or treble the consumption of oxygen for each 20°F. rise in tissue temperature. This chemical vasodilatation is not active at temperatures below 85°F., but increases rapidly at higher levels. As a result, if the supply of blood to the tissues is not correspondingly increased, the cells must suffer

oxygen want and this is invariably a cause of pain. Whereas the blood flow through the main pedal vessels is increased tremendously during the phase of acute hyperemia, there is good evidence that the cutaneous arterioles and capillaries have been injured and that therefore there may be a disparity between the supply and the demand for oxygen on the part of the tissue cells surrounding the cutaneous network of sensory nerve endings. On these physiologic grounds moderate cooling of the skin that is involved in a sterile inflammatory process will reduce the metabolic demand for oxygen. If demand can be made commensurate with supply, pain will be alleviated. Lewis[17] stated that spontaneous pain occurs in skin rendered hyperalgesic by heat and other types of injury, and that it is enhanced by warming and relieved or abolished by cooling. Both these factors constitute strong arguments for therapeutic cooling of hyperemic painful feet during the early stage of the post-immersion reaction. Actual experience has borne this out, as it has been found that reducing the cutaneous temperature to 80°F. gives consistent relief in the acute stage.

From a consideration of the above facts concerning the reaction of the tissues to cold, it becomes evident that the common denominator in all conditions that follow thermal insults is injury to the blood vessels and nerves. As Lake[8] pointed out, "a vicious circle is set up, for the greater the exudation the greater the venous obstruction, and the greater the venous obstruction the greater the rise in capillary pressure, and hence the greater the exudation."

Treatment

Prophylaxis

All ship's officers and seamen should be acquainted with the dangers of exposure to cold air and water. When there is sufficient time before abandoning ship, all hands should get into the most waterproof and loosely fitting boots they can obtain and take along extra pairs of heavy socks. If waterproof boots ship water and are wet inside, much can be accomplished by emptying them out periodically and putting on another pair of socks. This is the case even when the socks have been previously wet, provided they are wrung out and partially dried. Much can be done to prevent frostbite of the hands and feet by greasing the skin. Lewis[9] pointed out that the property of "supercooling" is abolished by wetting and washing the skin, but is greatly enhanced by greasing. A further point to remember is that men in crowded lifeboats must remain fairly immobile, with their legs dependent. Movement of the cold, benumbed extremities, even temporary elevation, and avoidance of constricting pressure on the legs from clothing or boat gear are helpful factors in preventing severe injury.

First-Aid

When shipwrecked survivors are picked up after prolonged exposure to wet and cold, they should be lifted aboard the rescuing vessel and carried below if their feet are numb, in order to prevent further injury to the anesthetic skin. The possibility of a severe reactive hyperemia and subsequent neuritic pain in the feet must be anticipated, even if actual gangrene from freezing has been avoided. These men require treatment for general as well as local chilling. Methods for dealing with the former are simple and well established, but while external and internal heat is being applied as a first-aid measure to the body as a whole, it should be borne in mind that the chilled extremities will suffer if they are warmed with equal rapidity. It is vitally important, therefore, to warm the patient as a whole first and prevent the chilled, bloodless extremity from increasing its metabolic demand for oxygen more rapidly than it can be supplied by a tardy restoration of the peripheral circulation. Although an appreciation of the risks of warming chilled extremities has been handed down as part of the folklore of the Eskimo, and the danger of this procedure was clearly understood by Larrey[3] over a century ago, it is not generally recognized by seamen today.

Whether a person has suffered actual freezing or the less severe chilling seen in immersion foot may not be at first apparent, but in either case the immediate local treatment is the same. It should consist in keeping the exposed extremity cool, elevated slightly above the level of the heart and surgically clean. There is no previous medical experience to point out the most effective methods of attaining these ends, and whatever methods are proposed here will undoubtedly be modified and improved. As local first-aid treatment on shipboard I suggest the following:

1. Give the skin of the feet a preliminary cleansing with soap and cool water. This is to remove superficial dirt and bacteria, which may cause infection of the blisters and thus open the way to cellulitis and moist gangrene. Surgical cleanliness should be scrupulously maintained until the blebs are resorbed and the danger of infection is past. If the blisters have ruptured, do not apply any antiseptics. Dust these raw areas with sulfanilamide powder or cover with sulfathiazole ointment, but if neither is available leave

them alone. On transferring these patients to a hospital ashore, the feet can best be protected against bacterial contamination from blankets and handling by covering them with a sterile towel or bandage (if available), or clean cotton socks.

2. Keep the feet cool by exposure to the room air. Have no bedclothes below the knees, the room cooled below 70°F., and, if possible, direct cool air from a blower or electric fan over the exposed feet and legs.

3. Avoid pressure points on the feet, especially the heels, and elevate the feet on a pillow. Under no circumstances permit these patients to sit up with their legs dependent, as this increases swelling. Elevate the legs so that the feet will be above the level of the heart and thereby promote drainage of edema fluid. Remember that anesthetic, swollen feet are vulnerable to trauma and infection. For this reason even gentle rubbing or massage is dangerous.

4. Use codeine or morphine as necessary to control pain.

Hospital

On arrival in the hospital, the severe cases have pronounced swelling, blebs and discoloration. They have the greatest amount of anesthesia and, for the first few days, the least pain. Hyperemia is striking, with dilatation of the main arteries and prominent veins, but circulation through the cutaneous arterioles and capillaries is nevertheless inadequate, as shown by a dusky cyanotic color and minute hemorrhages in the skin. As explained above, the rationale of treatment at this stage is to reduce tissue metabolism to a point where there is an adequate supply of oxygen to all the cells. This can be accomplished by cooling the skin 5 to 10°F. — to a point below 85°*. Experience has already shown that, under these conditions, color improves, blebs tend to resorb, and edema is reduced.† In the mild cases this degree of cooling can be brought about by simple exposure to room air, provided the ward temperature is kept below 70°F. Slightly greater chilling can be obtained by having an electric fan on a bedtable blow air over the feet (Fig. 4), and this can be rendered more effective still if the patient occasionally sprays water from an atomizer into the stream of air from the fan.

*Lake[9] deserves the credit for being the first to suggest treatment by continued cooling. In 1917 he wrote, "It would appear better to keep the limb cold by the application of wet cloths, until efficient treatment can be given, than to allow partial warming to occur." The Halifax experience has, however, demonstrated that wetting the skin is deleterious because it causes maceration.

†This is also true in an experimental burn. Drs. Oliver Cope and George Mixter, Jr.,[10] have shown that cooling the burned leg of a dog to 50°F. reduces edema formation to a striking degree, with a corresponding reduction in vascular extravasation and lymph production.

During the most intense stage of hot, congested feet, and also when the neuritic pains are severest, neither of the above measures is effective. The naval medical officers in Halifax have worked out a very satisfactory type of ice pack for use under these circumstances. This consists of a cotton hand

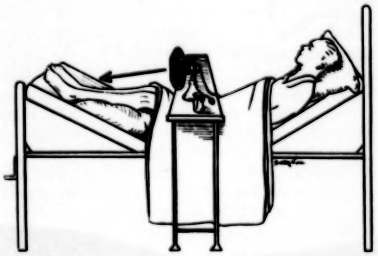

FIGURE 4. *Method of Cooling Feet by Exposure to Room Air Supplemented by Electric Fan.*

Note that the legs are elevated and that the heels are protected from pressure by a pillow under the calves. The injection of a fine spray of water into the revolving fan blades with an atomizer further increases the cooling.

towel to cover the foot; four ice bags applied over the dorsum, soles and sides of the foot; oil-silk wrappings to 12 cm. above the malleoli; a thick insulating pad of fluffed cotton or cellulose; and a rubber pillowcase tied loosely about the calf of the leg. The purpose of this pack is not to induce severe chilling, which might lead to further trophic disturbances, but to cool the skin of the foot and toes to a level between 80 and 85°F. During the early, intensely hyperemic stage a thermocouple inserted next to the skin shows that the temperature of the foot does not fall below 90°F., although the toe temperatures may drop 5 to 10° farther. At this period the active circulation causes rapid melting of the ice. The packs should be changed at four-hour intervals and observations of skin temperature made at these times to control their application. In Case 2 the feet maintained this intense hyperemia in continuous ice packs for the first eleven days, but on the twelfth day of treatment they became distinctly cool. The ice packs were then discontinued and treatment by exposure to cool air begun.

From this point on the patient can usually be made comfortable by simple exposure of his legs from the knees down, supplemented if necessary by a breeze from an electric fan as mentioned above. At night, however, the neuritic pains may increase in intensity. The patients appreciate the

value of the ice packs for relieving pain and often request their reapplication. Under these circumstances a less elaborate pack is usually sufficient, consisting of a cotton towel to cover the foot, two ice bags on the dorsal and plantar surfaces and a large bath-towel wrapping.

In carrying out treatment with ice packs it is important to make sure that they do not leak and wet the skin. Each time this has occurred it has caused sogginess and maceration.

In all the patients tested during the hyperemic stage, and even when it was subsiding, hanging the feet over the side of the bed caused striking congestion and pain. Conversely, elevation made the feet more comfortable. Many of the men discovered this for themselves and elevated their legs at the time of painful crises. The elevation need not exceed 20°. This is the best position for patients who are being treated by simple exposure of the legs to cool air, with or without an electric fan (Fig. 4).

On a theoretical basis, I advocate a period of Buerger's exercises prior to getting the patients out of bed. In these exercises, which are so useful in cases of threatened gangrene from arteriosclerosis or thromboangiitis obliterans, the feet are first hung over the side of the bed for four minutes, while the patient exercises the foot muscles by dorsiflexion and plantar flexion, inversion and eversion at the ankle and then flexion and extension of the toes. The feet are next elevated 45° for one minute, and then rested flat on the bed for five minutes. Cycles of these exercises carried out for half-hour periods three to four times a day should be tried as soon as the patient can tolerate the dependent position, and continued conscientiously for a week before permitting him to get out of bed.

Another measure often helpful in the early stage of edema and neuritic pain, particularly after prolonged exposure with starvation, is a diet high in protein and vitamin B. This is of specific value in survivors who have been adrift for long periods on a deficient diet, as will be shown in the second part of this paper.

On the basis of the similarity between the swelling due to immersion in cold water and that secondary to a burn, Koch's[19] method of compression dressings might be a valuable adjunct in treating patients with particularly severe edema and bleb formation, provided hyperesthesia of the skin is not a major complication. I hope to have the opportunity to give it a trial this coming winter. However, in view of the fact that thermal injuries from cold often have associated cutaneous anesthesia, care will have to be used in applying a tight dressing, in order to avoid trophic ulceration.

Operations for Relief of Pain and Vasospasm

Smithwick and White[20] have advocated crushing the peripheral nerves through small incisions above the ankle for the relief of intractable pain in cases of peripheral vascular disease. This operation has been extensively employed during the past twelve years and has proved valuable. When the nerves regenerate, as they do regularly in three months, they do so without pain. Such a procedure should also be of value in the control of the neuritic pain of immersion foot, provided cases occur with such severity that the methods given above prove inadequate. To test this out I injected the posterior tibial and the superficial and deep peroneal nerves in Case 10 with a 2 per cent solution of procaine and found that the patient obtained complete relief for a period of two hours. Nerve crushing should be reserved for the severe cases, as it will incapacitate the subject for duty for three months, the period required for full regeneration of the nerves.

Blocking the sympathetic outflow to the lower extremities by therapeutic paravertebral injections of procaine hydrochloride and by lumbar sympathectomy has been suggested by a number of French and American surgeons, notably Leriche,[21] Simon and Filhoulaud,[22] Soupault and Orsoni,[23] Forster and Wiederkehr[24] and Veal and Klepser.[25] I am unable to see how any possible improvement can result from such procedures during the acute stage. Absence of sweating proves that the terminal vasoconstrictor fibers are already interrupted, and the local hyperemia is more intense than that produced by sympathectomy. Indeed, the suggestions of Lewis and Love[26] and of Lake[8] that steps should be taken to constrict the main arteries seem more rational. As Greene[8] has stated, "it is to be hoped that without careful previous animal experiments no surgeons will be tempted to follow in the footsteps of these French colleagues." To see if the pain of immersion foot could be relieved by interruption of the sympathetic vasoconstrictors, I blocked the lumbar ganglions with procaine in Case 10. The patient's pain was not influenced in any way, but as I am not convinced that the sympathetic pathways were entirely interrupted, the test should be repeated. On the other hand, procaine injection of the posterior tibial and the superficial and deep peroneal nerves above the angle in the same patient abolished the pain completely.

The only case where lumbar sympathectomy is likely to be of value is in the late stages when the circulation of the foot remains insufficient. Four of the Halifax patients after recovery had unusual-

ly cold and sweaty hands as well as feet — a mild variety of Raynaud's disease. One of these (Case 19) who had had a Syme's amputation, continued to be incapacitated by ulceration at the end of the stump. He was tested by procaine block of the vasoconstrictor fibers (both by lumbar paravertebral and by subarachnoid spinal block), but showed no appreciable rise in cutaneous temperature in the ulcerated area. This was evidently due to organic narrowing of the peripheral vessels, secondary to his unusually prolonged chilling and septic gangrene. Nevertheless, cases will undoubtedly occur, as has already been observed in frostbite, where an inadequate circulation in the late stages of severe immersion foot (with secondary arterial occlusion) can be benefited by lumbar sympathetic ganglionectomy. A number of such patients, who six months after their initial exposure are still suffering from cold and pain in their feet, are under investigation at the United States Naval Hospital in Chelsea at the present moment. In addition to the inadequate circulation of their feet, the presence of increased sweating and of vasomotor changes in their hands indicates that they have an underlying vasomotor syndrome. Although it is unlikely that this condition originated with the exposure, it was probably increased by the psychic trauma.[27] Permanent vasodilatation may well result in secondary relief of the deep plantar pain.

Prognosis

Although it is too early to give accurate statistics of the end-results in immersion foot, it appears that the majority of patients respond favorably to the methods of treatment described above and should be able to return to duty within a month or six weeks. In the rare cases where gangrene develops or there is pre-existent peripheral vascular disease, and particularly when the sufferer has the misfortune to develop a psychoneurosis, convalescence may be very protracted.

Summary and Conclusions

Immersion foot, secondary to exposure to cold, appears to be caused by sublethal injury to the chilled tissue cells, the cutaneous arteriolar and capillary bed and the nerve fibers. The process differs only in the degree of chilling and wetting from frostbite, trench foot and shelter foot.

The ischemia during prolonged exposure to intense cold is followed by a period of acute inflammation. The hyperemic reaction of the feet is due mainly to the chemical effects of the products of tissue injury. Paralysis of the vasoconstrictor fibers is an added factor, but is of secondary importance.

In spite of the increased flow of blood in the main vessels, there is a disparity between the blood-oxygen supply and demand in the cutaneous capillary bed. Capillary permeability is thereby increased. Edema, pain and petechial hemorrhages, as well as the congestion seen when the feet are in the dependent position, are evidence of injury to the blood vessels. By cooling and reducing the local metabolism and need for oxygen, both edema and pain are diminished.

The inflammatory reaction and hyperemia fade in a few weeks, and the circulation usually returns to normal at this time.

Neuritic pain makes its appearance with partial recovery of the nerves and continues until nerve function is restored. It has disappeared within two weeks in some of the mild cases seen in Halifax, but it is impossible as yet to tell how long it may last in the severe ones. In some of the survivors observed in Scotland and in this series, it was troublesome for nearly six months.

Much can be accomplished by suitable treatment. This must be directed primarily at reducing local tissue metabolism in the early period and in preventing further injury and infection of the insensitive swollen feet. Gangrene should be a rare complication.

The rationale and methods of treatment developed so far are outlined, with suggestions for future investigation.

Not all cases of swollen painful feet seen in shipwrecked survivors are due to immersion in cold water. Cases in which this condition has developed in a tropical climate will be discussed in the second part of this paper.

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FIEDLER'S MYOCARDITIS*

Report of a Case

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NEW ORLEANS, LOUISIANA

SINCE Fiedler[1] first described interstitial myocarditis in 1900, there have been to our knowledge 9 reported cases of this entity in the American literature. Attention was first directed to this unusual form of myocarditis in the United States by Scott and Saphir.[2] In the world literature we were able to find at least 46 cases that conform to the pathologic criteria consistent with this syndrome.

Many quasi-descriptive terms have been used to identify the condition. Acute interstitial myocarditis was proposed by Fiedler. Later other synonyms were applied, and terms such as acute isolated interstitial myocarditis[3]; acute, subacute and chronic isolated myocarditis[4,5]; myocarditis perniciosa[6] and granulomatous myocarditis[7] have appeared in reviews and case reports of this condition.

Because of the rarity of this syndrome, we believe that a brief survey of the literature and the report of an additional case are justified.

Case Report

L. E., a 29-year-old housewife, was admitted to the Tulane Medical Service on October 21, 1939. She had been perfectly well until August of that year, when she experienced a sudden attack of "smothering" while doing her daily housework. At this time she was conscious of her heart's palpitating and was unable to get her breath. In a few minutes she was able to resume her work. Following this attack, she had occasional periods of dyspnea on exertion, which became progressively greater. Nine weeks prior to admission she began to experience attacks of paroxysmal nocturnal dyspnea. At about this time she began to require two to three pillows at night and developed a cough productive of frothy sputum. Her activity was greatly restricted. Despite this the attacks of dyspnea became more frequent. On October 11, 1939, the patient was ordered to bed by her physician. At about this time she began to develop upper abdominal pain, noticed swelling of the upper abdomen, and began to vomit bile-stained material. Because of her failure to improve, the patient was referred to the Charity Hospital.

The past history was relevant in so far as it showed that the patient had had a cholecystectomy for cholecystitis and cholelithiasis 3 years previously. She had had "kidney trouble" and "high blood pressure" with a pregnancy 7 years previously. Subsequent follow-up over a period of years revealed no hypertension.

Physical examination disclosed an obese white woman who appeared to be of her stated age. The temperature was 100.4°F., the blood pressure 120/80, the respirations 30 and somewhat labored, and the pulse 108. There was enlargement of the right lobe of the thyroid gland, which was firm and smooth. Examination of the chest revealed lagging on the right side. The point of maximal cardiac impulse was felt in the 6th left interspace 8 cm. from the midsternal line. Tactile fremitus was diminished on the right posteriorly. There was dullness to percussion in this area below the 4th interspace. There was no enlargement of the heart to the right. The left border of cardiac dullness was 9 cm. from the sternal line in the 6th interspace. Breath sounds were heard with difficulty at the bases of the lungs, and vocal fremitus was greater on the left than on the right. Auscultation of the heart revealed no murmurs. The remainder of the physical examination was not noteworthy.

The red-cell count was 5,250,000, the hemoglobin 70 per cent, and the white-cell blood count 15,000, with a normal differential. The sedimentation rate was 20 mm. in 1 hour. Urinalysis was not significant, the specific gravity being reported as 1.022. The blood Wassermann reaction was negative. A roentgenogram of the chest was reported as showing enlargement of the cardiac shadow with evidence of pulmonary congestion. There was evidence of some fluid in the right pleural cavity.

The patient was digitalized, given oxygen by nasal catheter, and a thoracentesis of the right chest on October 22 yielded 1000 cc. of straw-colored fluid. Two days

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after admission a gallop rhythm and frequent ectopic beats were heard. A few days later the temperature rose to 100.2°F. and the patient ran an irregular fever for 9 days, no cause other than pulmonary congestion being found.

The basal metabolism was as follows: on November 8 the rate was +23 per cent, on November 10, +13 per cent, and on November 15, +3 per cent. Electrocardiograms made on October 23 revealed occasional ventricular premature beats and low, bizarre QRS complexes. T_2 was diphasic, and there was depression of the RST segment in Leads 1 and 2. The interpretation of the electrocardiographic evidence was extensive myocardial disease.

On November 25 the patient left the hospital against advice.

After leaving the hospital the patient felt quite well until June, 1940, when she began to decompensate. In the interval between June and November she was admitted to the hospital on two occasions. Her fourth admission occurred on November 25, 1940, when she returned at the request of the staff for further observation. At that time the blood pressure was 110/68. Investigation revealed that the heart had undergone progressive enlargement, particularly to the left. This was confirmed by roentgenologic examination.

On August 12, 1941, the patient was readmitted for the fifth time. She had been taking digitalis regularly up to 1 month before admission, when, on the advice of her physician, it was stopped for 14 days. In 2 weeks she developed dyspnea, palpitation and edema. Digitalis was resumed, but because of her heart failure she was advised to return to the hospital.

At the time of this readmission the temperature was 99°F., the pulse 90, and the blood pressure 130/80. Congestive rales could be heard at both bases up to the mid-lung fields. The heart border was percussed 4 cm. to the right of the sternal line and 13 cm. to the left of the midline in the 6th interspace. A gallop rhythm was heard. There were frequent ectopic beats and at times bigeminy was thought to be present. A pulse alternation of 10 mm. of mercury was noted. A systolic mitral murmur was heard. The liver was palpable three fingerbreadths below the costal margin. Moderate edema of the feet, legs, sacrum and hips was present. The venous pressure was 24.8 cm., and the Decholin circulation time was 45 seconds. The patient was redigitalized and slowly improved, and early in September was allowed out of bed for short intervals. On September 21, she began to feel nauseated, vomited, and complained of abdominal pain. Because she was theoretically digitalized or perhaps overdigitalized, the drug was discontinued. She rapidly became worse, complained of dyspnea, and digitalis was resumed on September 24, with little effect. On September 28 the patient became extremely dyspneic and oxygen was ordered. She expired at 3:30 p.m. on September 29.

Autopsy. The outstanding pathologic changes were present in the heart, which weighed 450 gm. The epicardium was smooth and shiny, and the myocardium was relatively flabby. The coronaries were widely patent. In the region of the apex in the left ventricle there was a brownish, softened area that shrank beneath the cut surface and measured 0.8 cm. in diameter. In the inferior region of the left ventricle there was a firm, whitened scar penetrating a columna carnea that was approximately 1 cm. in length. The mitral valve had a number of wartlike protuberances, which appeared to be of the arteriosclerotic type. They were much larger than rheumatic lesions, being approximately 0.4 cm. in diameter. The auricular endocardium above the mitral valve was thrown into threadlike folds and projections. Measurements were as follows: tricuspid valve, 13.0 cm.; pulmonary valve, 7.4 cm.; mitral valve, 10.7 cm.; aortic valve, 6.0 cm.; right ventricle, 0.5 cm.; and left ventricle, 1.6 cm. The

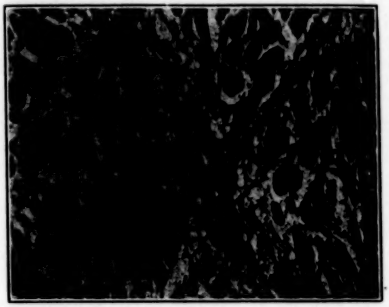

FIGURE 1.

columnae carneae of the left ventricle were filled with grayish, apparently organized but easily removable thrombi. The other findings were pulmonary edema and congestion, chronic passive congestion of the liver and spleen and follicular cysts of the ovary. The thyroid gland showed no evidence of disease.

Microscopic examination of the heart revealed many areas of inflammation in the myocardium, varying in de-

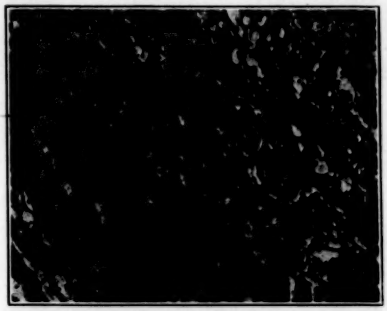

FIGURE 2.

gree and character. There was evidence of injury and repair of the muscle fibers associated with an infiltration consisting chiefly of lymphoid cells and an occasional polymorphonuclear leukocyte and phagocytic cell. Small areas of interstitial hemorrhage were present, but these were not remarkably prominent. There were areas where the degree of hydrops of the muscle was extreme (Fig. 1). At other sites an older lesion was demonstrable in which fibroblastic proliferation dominated the picture (Fig. 2).

Discussion

The history and personal observations of the course of this case lead us to conclude that the disease began in June, 1939, and progressed to the patient's death in September, 1941. Search of the available literature on this subject discloses no other case of a patient with Fiedler's myocarditis surviving more than twenty-one months.

Many authors have attempted to incriminate definite etiologic factors that have been observed in connection with this disease. In the individual case reports carbuncles, gonorrheal urethritis, upper-respiratory infection, influenza, "rheumatism," syphilis and status thymicolymphaticus have been described as being associated in patients who have been found at necropy to have interstitial myocarditis. Such an association is believed by most authorities to be coincidental and as yet no definite etiologic relations have been established.

Clinically no constant progression of symptoms and signs that is diagnostic of this pathologic entity is known. Its manifestations are protean. The reported cases show no preponderance of either sex. The disease occurs primarily between the ages of eighteen and fifty, although cases have been observed in patients as young as nineteen months[8, 9] and as old as sixty-nine years. The onset in the majority of cases is abrupt and may be accompanied by a chill. It may, however, be initiated by upper respiratory symptoms, such as cough, dyspnea, hemoptysis, generalized weakness and precordial distress.

Physical examination usually reveals no findings of any consequence. Some cases have presented evidences of focal infection of varying severity. There has been in some cases a slight but definite elevation of temperature for which no explanation was evident. In general the heart is enlarged, predominantly to the left, and at times a soft apical systolic murmur is heard. The rate is invariably accelerated, and a gallop rhythm has been described. There have been comparatively few electrocardiographic studies, but most of those reported presented evidence of definite myocardial damage. Despite this no diagnostic electrocardiographic criteria have been established for the condition.

The duration of the disease has been found to range from two days[10] to twenty-one months.[5] In fact, one case is reported[10] in which a patient died suddenly from a rupture of the left ventricle four days after a convulsive seizure. In the usual case, however, the disease will have run its course after a few months.

The histopathologic picture of this entity is characterized by typical findings.[11] There is usually an interstitial infiltration of the myocardium, which is chiefly due to lymphocytes, monocytes and, to a certain extent, neutrophils, eosinophils and plasma cells. Areas of necrosis are not infrequently found in the muscle fibers. This injury is diffuse throughout the myocardium. In some cases, hemorrhages have been described, due in all probability to the rupture of hyalinized muscle fibers. In the more chronic cases these areas are replaced by acellular fibrous scars. There may be actual granulation tissue with new capillary formation and fibroblastic proliferation.

Covey[12] emphasizes that no descriptions or illustrations contain all the abnormalities of the aggregate but that the variations pictured in the different case reports are insignificant when the whole is analyzed. It appears that whatever the etiologic agent may be, there is nothing in the histologic picture suggestive of a specific granuloma.

Summary

A case of Fiedler's myocarditis is reported, and the significant clinical and pathological findings, as reported in the literature, are discussed.

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MEDICAL PROGRESS

RESPIRATORY FAILURE IN ACUTE POLIOMYELITIS

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BOSTON

THE increased interest in the treatment of acute poliomyelitis awakened by the Kenny technic has resulted in a certain amount of confusion regarding the value of the respirator in the presence of respiratory failure in this disease. The Kenny school of thought has gone so far in its condemnation of "orthodox practices" in this disease as to advocate the abandonment of the respirator in favor of the application of hot packs to respiratory muscles. This willingness to scrap the respirators along with the immobilizing splints calls for a review of the many complex factors involved in respiratory failure in acute poliomyelitis.

The muscles of respiration consist of two groups, one of which is engaged in quiet breathing known as eupnea; the other group, composed of the accessory muscles of respiration, comes into play during labored breathing or dyspnea. In the first group, there is a variation in the part played by the different muscles according to whether the subject uses the abdominal or costal type of respiration. In the dyspnea met with in the spinal type of poliomyelitis, the exaggeration of the breathing effort is manifested in those muscles that are attempting to compensate for muscles either weakened or completely thrown out of commission by spasm or paralysis. A muscle or a group of muscles that is in spasm may not be completely lost so far as potential function is concerned. In fact, muscles with an uninjured nerve supply may be thrown into continued contraction by the spasm of opposing muscles,[1] the so-called "brake" phenomenon in Kenny terminology.[2] Thus it is that the number of respiratory muscles thrown out of commission may be out of all proportion to the actual number of muscles or muscle fibers directly affected by nerve damage. This lost function is compensated for by the increased effort of the remaining, freely active muscles. This effort varies not only according to the amount of function lost but also according to which muscles are affected. There may be exaggerated abdominal breathing to compensate for loss of function in the thoracic group, or exaggerated thoracic breathing to compensate for loss of function in the abdominal group. But as the demand for compensation increases, the accessory muscles of respiration come more and more into activity in an effort to lengthen and expand, as well as to contract, the thorax.

ANATOMY

The muscles engaged in forced inspiration are numerous. First of all is the diaphragm, which on contraction pulls air into the chest and simultaneously pushes out the abdominal wall. The thorax is also elongated by the upward pull of the three pairs of scaleni on the first and second ribs. The sternocleidomastoid and trapezius serve as accessories in this accordion process. Expansion of the thorax is accomplished by contraction of the levatores costarum, the external intercostals and the serratus posticus superior. By their action the ribs are raised and the shafts slightly rotated outward. In addition to all these we have the accessory action of the rhomboidei, pectoralis major and minor, serratus magnus, latissimus dorsi and, finally, the extensors of the spine.

Forced expiration is accomplished by contraction of the abdominal muscles, which, with the glottis open, forces the dome of the diaphragm upward and outward. Engaged in this process of shortening the thoracic cavity are the external oblique, internal oblique, transversalis and rectus, whereas the internal intercostals, triangularis sterni, transversus thoracis and serratus posticus inferior tend to contract the thorax.

Although the function of some of these muscles is open to debate, they nevertheless all come into play in dyspnea. It is worthy of note that some one hundred and twenty separate muscles, as enumerated above, may go into vigorous action in forced respiration. Furthermore, it is clear that many of these, including the diaphragm, are far removed from any direct influence of hot packs. However, one can imagine that if the external intercostals were thrown into spasm and if the internal intercostals were to go into spasm by reflex action, both sets might be benefited by the application of hot packs to the chest wall. In this connection, it is a matter of clinical experience

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in poliomyelitis that the muscles of expiration show signs of serious fatigue sooner than do the muscles of inspiration. This coincides with the fact that even normal inspiration requires more muscular work than does expiration. The Drinker and the Emerson respirators, by creating rhythmic negative pressure, take over the work of the inspiratory muscles. The pulsator of Bragg and Paul, on the other hand, rhythmically compresses the chest wall by pressure from a rubber bag fastened about the chest. This compression takes the place of the expiratory muscles in forced expiration, as in the Schaefer method of resuscitation. Of the two, the respirator much more closely imitates normal respiration.

Physiology

The respiratory center in the medulla or bulb lies under the floor of the fourth ventricle in the region where the cranial nerves originate. This center has a right and a left half, which communicate with one another through the intrabulbar commissural fibers, giving a symmetry of respiratory movements. The main function of this center is to regulate the rhythm and the depth of the involuntary respiratory movements. It does this through afferent impulses arising from various sources which convey the body needs. From here also fibers pass directly downward without decussation to the respiratory motor nuclei in the anterior horns of the cord. Any impairment of this respiratory center in the medulla results in variations in rhythm and depth, and its destruction results in immediate cessation of respiration.

This respiratory center is in anatomic and physiologic relation to certain cranial nerves whose afferent fibers bring controlling impulses of inspiration and expiration. The most important of these is the vagus, the sensory fibers of which come from the lungs, bronchi, trachea and larynx, whereas the motor fibers supply these same areas, together with the pharynx and soft palate. These impulses exert essential influences for both the rate and the rhythm of involuntary respiration, and also for the more violent voluntary efforts of coughing.

Sensory fibers of the trigeminal nerve arising from the nasal mucous membrane have an important protective function. Any violent irritation of these nerve terminals by injurious gases inhibits breathing. The same effect is obtained from any marked irritation of the glossopharyngeal nerve. In fact, the sensory fibers of this nerve inhibit respiration whenever one swallows.

Involuntary respiration is under the control of the respiratory center in the bulb. Voluntary control comes from the cerebrum, as in talking, singing, whistling and any blowing effort. But there are impulses that involuntarily interrupt the normal rhythm, such as yawning, laughing, sighing, crying and sobbing. Coughing and sneezing arise from irritation along the tract. Hiccup, a spasm of the diaphragm with closure of the glottis in the form of an extra inspiratory wave, can be induced by a wide variety of causes including irritation of the diaphragm, intoxication, encephalitis, irritation in the trapezius[3] and exsanguination in its terminal stage. Of these all but the last may occur in poliomyelitis. The hiccup in this disease is a subject that in itself deserves extensive discussion.

Spinal Respiratory Failure

When a healthy individual is placed in a respirator, his respiratory rhythm is promptly taken over by the machine without his becoming aware of it until he tries to talk. He can articulate only with each expiration. He finds himself merely making motions of the tongue and lips during inspiration, and he cannot prolong expiration against the machine. A patient in the respirator learns very promptly—within a few minutes—how to talk and how to swallow nourishment, if minute amounts are given at first. He does not have to be taught by anyone: it is a matter of extraordinarily prompt adaptation to an artificial respiration that closely simulates normal breathing.

The respiratory center serves as the pacemaker for a wave of musculature contraction starting in the diaphragm and co-ordinately bringing into play those muscles of the thorax and abdomen that are needed. The diaphragm may "flutter" in post-encephalitic conditions and, in so doing, may carry the thoracic muscles along at the rate of 90 or even 200 contractions a minute, a very uneconomical form of respiration, with movement of air largely confined to the "dead spaces," and with very little change of air in the lung itself. Dowman[4] and Skillern[5] have shown that under these conditions, when the diaphragm is put out of commission by a double phrenectomy, the other respiratory muscles promptly return to a contraction rate of 20 per minute. Gamble, Pepper and Muller[6] observed that occasionally in flutter the chest muscles would "miss" and, further, that immediately after prolonged holding of the breath or during sleep the diaphragmatic flutter would at times fall into an exact two-to-one rhythm with the thoracic excursions.

All this puts one in mind of the relation of the auricles to the ventricles in cardiac rhythm, an analogy discussed by Wenckebach.[7] But there is a wide difference between the respiratory and the cardiac systems, since there is no pacemaker in the diaphragm and no "conduction system" other than the nerve supply from the respiratory center to the

respiratory muscles with the synapses in the spinal cord. Nevertheless, the co-ordination in the wave of contractions and the disturbances in rhythm offer an interesting comparison from a clinical viewpoint. An appreciation of these outward similarities helps one to recognize some of the respiratory phenomena observed in poliomyelitis.

In spinal poliomyelitis only one side of the diaphragm may be thrown out of commission.[8] One may observe a patient getting along fairly satisfactorily with apparently a complete unilateral absence of contractions. Likewise a patient will sometimes appear to be getting along well with only the diaphragm and very little use of either the thoracic or the abdominal muscles. However, these patients should always be in close proximity to a respirator, and be put in at the first sign of fatigue. Although a patient may seem to be getting enough air under these conditions, he will often get relaxation and comfort in the respirator so that he promptly falls asleep.

Indications of impending failure in the spinal type of poliomyelitis consist of visible loss of action in the muscles of normal inspiration, with overaction of the sternocleidomastoid ("neck breathing") and other accessory muscles. The breathing is at first shallow and later labored. Speech is interrupted by inspirations so that the patient can count only to two or three before breathing in. Coughing becomes ineffective and then impossible. Cyanosis implies anoxemia, with lowered resistance to the infection as a whole, as well as loss of muscle strength. When all inspiratory effort is apparently on the point of exhaustion and the expiratory muscles appear to be attempting to compensate with a low vital capacity of air in the lungs, the need of a respirator is imperative. The ideal time to put the patient in is at the first signs of failure. Delay is to his distinct disadvantage.

The supportive value of the respirator when early fatigue is threatened is difficult to determine in statistics, yet it may be quite obvious to the patient and to the attending clinician. Nor does such a statement preclude the use of hot packs as an intermediate measure. Indeed, I have seen hot packs enhance the inspiratory effort of the thoracic muscles when these were lagging, but such benefit was temporary. One may assume that muscular spasm is relaxed by the heat, but certainly the heat does not reach a diaphragm in spasm. Whether the respirator has a salutary or detrimental influence on any of the respiratory muscles in spasm is unknown. At least, one can say that there is no danger of muscle-stretching. Short of complete paralysis there is certainly no rest for these muscles, in the true sense of the term, while life goes on. The respirator may secure rest to some of the overworked accessory muscles, but primarily it serves as a support to the muscles of normal quiet inspiration.

As pointed out by Wilson,[9] the nearest approach to achieving rest for the respiratory muscles comes from the proper use of a respirator. This machine is to failure of the respiratory muscles what intubation is to laryngeal obstruction. In this comparison the preliminary trial of hot packs may be compared to the use of steam inhalations in laryngeal spasm. There are definite indications for the respirator as there are for intubation. Delay in the use of the respirator as with the tube results in fatigue and anoxemia, and may mean the difference between death and recovery. Just as the tube should not remain in any longer than is necessary, so also the patient should be weaned from the respirator as soon as possible. Ability to cough is a good indication for removal.[10] The respiratory muscles can be "alienated" from the control of the respiratory center as definitely as the leg muscles can be "alienated" from the cortex by prolonged immobilization in a splint.

In complete and irreversible paralysis of the respiratory muscles, life can be maintained only by the continued use of the respirator. However, as pointed out by Wilson,[11] there is no way of telling at the beginning of muscular failure how much nerve damage is actually taking place. Nor, as he says, is there any room for philosophical discussion of one's duty as a physician even if one could foresee indefinite dependence on the respirator. Furthermore, the established fact that there is a high death rate from respiratory infections following the use of the respirator is also no argument against its use, since this also holds true after intubation and tracheotomy. Indeed, the utmost care should be used in respirator cases to avoid exposure to respiratory infections. All attendants should wear masks, and no visitor with a cold or sore throat should ever be allowed in the room.

The rate of the machine is not so important as the height of negative pressure. In the pure spinal type the patient's respiratory rate depends on the degree of failure, but once in the apparatus he promptly adapts himself to its rate. This is particularly shown in the room respirator where several patients are being treated at the same time.[12] The rate for children is usually between 20 and 30 per minute, and for adults between 15 and 20 per minute. The pressure, however, is a very different thing. The real danger of the machine lies here. Too great a negative pressure injures the alveoli of the lungs and thereby induces pneumonia.[10] Children under ten years of age should be started at a pressure of 10 cm. on the water manometer, and this should never be allowed to

rise above 14. Adults can be started at a pressure of 12 to 14 cm., and there is rarely any reason for allowing it to rise above 16, and never above 20. The four most important points to keep in mind in the use of the respirator in the spinal type of failure are these: avoid delay; wean as early as possible; avoid dangerously high negative pressure; prevent bedsores. The nursing care of respirator cases is described in a brochure put out by the National Foundation for Infantile Paralysis.[13]

Bulbar Respiratory Failure

Respiratory paralysis in poliomyelitis is not confined to impairment of the respiratory muscles through injury to their spinal-nerve supply. The virus may attack other portions of the nervous system whereby respiration is affected, either directly at the respiratory center itself, or indirectly through injury to those cranial nerves supplying certain muscles that clear the respiratory tract by swallowing.

When the respiratory center itself becomes impaired, the respiration becomes irregular and jerky. This irregularity is not overcome by the respirator. In other words, the respiratory movements fail to synchronize with the machine. Along with this there may be long sighs or violent hiccups that add to the confusion of the breathing. In a way this may be compared to the twitching and fibrillation seen in the trapezius muscle when its anterior-horn cells are being invaded by the virus. Suddenly and without further warning respiration stops. On the other hand, one may see a marked slowing of the rate with deep inspirations, signifying pressure on the floor of the fourth ventricle from increased spinal-fluid pressure. Lumbar drainage promptly performed can offset such a situation, and with repetitions as needed may lead to the return of normal respirations as the pressure subsides.[14]

The function of deglutition is vital to respiration, as this keeps the upper respiratory tract clear. Any impairment of this function through injury to the cranial nerves supplying this area results in choking and finally drowning in mucus. This is the so-called "wet type" of respiratory failure. To speak of these patients as dying of pneumonia is misleading. Of course, they may do so later from a secondary infection. In these cases with inability to swallow the respirations are shallow and often irregular, because the patient hesitates to breathe for fear of inspirating mucus into the trachea.[15] With laryngeal paralysis coughing becomes impaired because of an inability to close the glottis. Thus it becomes impossible to clear the throat by either swallowing or coughing. The accumulation of mucus tickles the underlying mucous membrane, with the result that the secretion of mucus is rapidly augmented. Nausea may be an additional factor in the promotion of excessive mucus. It is possible that the invasion of the pharynx by the virus of poliomyelitis in these cases may also be another factor in this profuse secretion of mucus. The recent work of Sabin[16] indicates that bulbar invasion originates from extension of the virus along the neurons supplying the pharynx, whereas spinal involvement originates from extension of the virus along the neurons supplying the intestine.

Landon and Smith,[17] Kelleher[18] and Emil Smith[19] state that atropine is useful in drying up these secretions. Crone[19] remarks that atropine gives only temporary and partial relief. Wilson[20] has found it unsatisfactory and believes that it may render the mucus more tenacious, more annoying to the patient and more difficult to remove. My own experience is that, although atropine is oftener useless than helpful, it has at times appeared to slow up the secretions perceptibly.

Suction applied to the pharynx is a great help, but it must be exerted very gently. Anderson[21] uses a rubber catheter with numerous openings near the tip. This is passed through one nostril down into the pharynx and constant suction is applied. With sufficient skill a tube may be passed through the other nostril and down through the esophagus to the stomach for the purpose of feeding. Needless to say, one must be sure that this tube lies in the stomach. The foot of the bed is raised sufficiently to give the trachea enough of a downward slant to procure gravity drainage.

Postural drainage is of far greater importance than either atropine or suction, as has been graphically brought out by the cases reported by Durand.[22] With the foot of the bed raised 2 feet enormous quantities of this mucus—a pint or more—will run from the nose and mouth in spite of the administration of atropine. The patient's position can be shifted from one side to the other—better still with the face down but turned to the side.

The respirator is not only useless for correcting the embarrassment to respiration in bulbar poliomyelitis, but can be actually harmful. As has already been said, when the respiratory center is involved the patient's breathing does not synchronize with the machine. Thus the machine interferes with his breathing instead of supporting it, and he begs to be taken out. When cranial-nerve involvement causes pharyngeal paralysis, the respirator causes the mucus to be forcibly drawn down into the trachea, whereas the patient's own intuition prompts him to avoid this by shallow breathing. Nevertheless, it must be clearly

understood that a patient may have a combination of spinal and bulbar involvement, and the spinal involvement may be sufficiently severe to warrant a trial of the respirator, provided precautions are taken against the effects of the bulbar paralysis. Especially is this true where the spinal involvement is severe and the bulbar involvement is mild. When pharyngeal paralysis is combined with the spinal type, an inlaid catheter with constant suction can be tried along with the respirator. To meet this combination of circumstances a respirator is now available that can be adjusted like an operating table and thus afford postural drainage as well. However, the mortality rate in these cases is always high, especially in those with the ascending type of paralysis.

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There is one condition that occurs, especially in the ascending type of spinal paralysis, where the respirator becomes useless. The abdominal muscles become paralyzed first, as their nerve supply comes from the dorsal segments of the cord. Next, the thoracic muscles cease to function, their nerve supply coming from the cervical segments. If the diaphragm, which is also supplied by the cervical segments, holds out long enough to get the patient to a respirator, dramatic and immediate relief may be obtained. But unfortunately, the paralysis of the intestinal tract then begins to manifest itself with absolute stasis and increasing abdominal distention. Hot stupes, enemas and the rectal tube become ineffectual. A Miller-Abbott tube cannot be worked down beyond the pylorus under these conditions. The weakened and eventually paralyzed diaphragm is forced higher and higher. Finally the negative pressure of the respirator, even when increased to 30 cm. on the water manometer, fails to enlarge this contracted thoracic cage. A respiratory standstill may thus ensue in spite of the respirator before the medulla is encroached on.

Condemnation of the respirator by the close adherents of the Kenny method has been uttered from the lecture platform. I witnessed this in the crowded Kenny booth at the last meeting of the American Medical Association in Atlantic City, and again at the course given at the University of Minnesota last July, where the lecture and motion picture dealing with respiratory paralysis were unforgivably narrow in scope and in tone. This exercise was not conducted by Sister Kenny. In her book Sister Kenny[2] describes the success of her treatment in two early "respirator cases" in which the patients were "unable to swallow," and in a spinal case where artificial respiration by "gently depressing the floating ribs" was resorted to five days after the onset of the poliomyelitis. She describes it as a form of "muscle education"— that is, teaching the patient to breathe. That Sister Kenny inspires confidence in her patients and overcomes their fears is one of her great assets. But one is also impressed by her outspoken prejudice against all orthodox treatment. Thus, she prefers the old method of resuscitation that all physicians had to use before the invention of the respirator. A careful analysis of the respiratory cases treated by Daly and her associates[23] by the Kenny method is not at all impressive in this particular respect, although otherwise it is highly instructive. Enthusiasm for the Kenny technic has led many to accept it as an ell-embracing system of treatment for poliomyelitis. Such an attitude is out of keeping with the professed philosophy of Sister Kenny herself at her highest moments as a teacher. Hot packs to the chest, neck and abdomen may be advantageously applied in the earliest stage prior to insertion in the respirator and on removal from it, or where no respirator is available.

The use of the respirator is fraught with many discouraging experiences. Some are due to the types and complications of the respiratory failure, but others result from the overwhelming damage brought about by the progress of the disease. These experiences have been stressed by all authors, and powerfully expressed in an editorial.[24] Nevertheless, on the credit side there are an ample number of patients who have obtained comfort, relaxation and sleep at a critical period of respiratory embarrassment, and who later have walked out of the hospital to lead useful lives. It is impossible to draw up an adequate protocol of the results of respirator treatment over the last decade, because of the relatively small number of publications on this subject. However, in a survey of the year 1940 Wilson[24] found that among 127 cases with simple intercostal or diaphragmatic paralysis treated in the respirator, 81 per cent survived. The survey could not include the end-results regarding permanent handicaps. It did bring out that in general the respirator was too frequently used where it was not indicated, and that where indicated its use had too often been delayed to the distinct disadvantage of the patient. Unqualified disparaging statements regarding the value of the respirator increase these delays. The Kenny method has much in its favor, but as yet no figures have been produced to show that hot packs can replace the position now held by the respirator in the treatment of the spinal type of respiratory failure in poliomyelitis.

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CASE 29071

Presentation of Case

A sixty-five-year-old woman entered the hospital because of "gas around the heart" and palpitation.

Approximately one year before entry the patient developed slight constipation, which was relieved readily with mineral oil or milk of magnesia. Since that time she had had intermittent attacks, that is, two or three times per month, of periumbilical crampy pain, each lasting several hours. These were unrelated to food intake or activity and were sufficiently severe on only one occasion to force her to bed. She had had no recurrence of this pain during the four or five weeks prior to admission. She attributed this to pills prescribed by her physician. However, five weeks before entry she began to suffer with considerable belching and sour eructations. The belching was worse at night and half an hour after meals, but was not otherwise related to activity and was not effected by bowel movements. During this same period, palpitation frequently occurred at night. At no time had there been malaise, anorexia, nausea, vomiting, diarrhea, melena, jaundice, dyspnea, orthopnea or ankle edema.

The family and past histories were noncontributory.

Physical examination revealed a moderately obese, somewhat apprehensive woman who did not appear to be uncomfortable. The chest was barrel shaped. Examination of the lungs was negative. The heart was considered enlarged to the left; the sounds were of good quality and no murmurs were audible. The abdomen was soft. There was slight tenderness and a poorly defined mass in the left lower quadrant.

The blood pressure was 167 systolic, 90 diastolic. The temperature was 97.6°F., the pulse 88, and the respirations 18.

The examination of the blood revealed a hemoglobin of 14.7 gm. and a white-cell count of 9700 with 69 per cent polymorphonuclear leukocytes. The urine was normal except for a rare white and epithelial cell in the sediment. A gastric analysis revealed no free acid in the first specimen and 15 units in the specimen collected an hour later. The gastric contents were guaiac negative. Repeated stool examinations were guaiac negative. A Graham test was negative. A barium enema showed narrowing and marked mucosal swelling of the midportion of the sigmoid over a distance of approximately 10 cm. Definite ulceration of the mucosa was not visible. There were numerous diverticulums in the sigmoid and descending colon. The colon proximal to the lesion was not examined. Ten days later the barium studies were repeated, barium being introduced by means of a Foley catheter. At this examination there was complete obstruction in the sigmoid in the area of greatest narrowing previously described. The exact nature of the lesion could not be demonstrated because insufficient barium passed the point of obstruction.

An operation was performed on the sixteenth hospital day.

Differential Diagnosis

Dr. Reginald M. Smithwick: The history indicates that this sixty-five-year-old woman had had symptoms referable to the gastrointestinal tract for about a year. One of these was slight constipation, which apparently did not increase in severity and was easily relieved by nondrastic laxatives. There were also short periods of crampy abdominal pain occasionally lasting several hours. Later, belching and sour eructations but no nausea or vomiting were noted. There was no distention, increased peristalsis or fever. An indefinite, slightly tender mass in the left lower quadrant tends to incriminate the large intestine. This was confirmed by a barium enema, which revealed a process involving the midportion of the sigmoid — with marked narrowing on the first examination and complete obstruction on a second examination ten days later. There is no evidence in the history of other clinical signs of complete obstruction, such as distention, pain, increased peristalsis, nausea and vomiting. Apparently her bowels moved in spite of the x-ray evidence of marked obstruction, since it is reported that repeated stool examinations were negative for occult blood.

The question for discussion seems to be the nature of the lesion in the sigmoid colon. The x-ray suggests an intrinsic process. A review of the films may be helpful.

Dr. George W. Holmes: There is nothing of interest in the chest. The gall bladder is well seen and looks normal. The colon is obviously abnormal, and the picture is characteristic of diverticulosis, with multiple large diverticulums scattered throughout the descending colon and sigmoid.

At one examination there was no definite obstruction. At another examination ten days later there was definite obstruction. At the first examination, in addition to the diverticulums, there was evidence of spasm. The film taken in the lateral view shows a good outline of the lumen of the large bowel, and it does not seem possible that a tumor large enough to plug the bowel could have been present and have been completely missed at this first examination. At the second examination the filling is insufficient and one could miss a tumor, but I should not expect a large tumor to appear in such a short interval. So far as the x-ray demonstration goes there was no ulcerated lesion or tumor, so our interpretation would be that the obstruction was due to the diverticulitis.

Dr. Smithwick: The two most likely diagnoses are carcinoma and diverticulitis, with or without a localized perforation and abscess. It is possible that both lesions were present. These seem more probable explanations for the sigmoid lesion than tuberculosis, lymphoma, endometriosis, metastatic carcinoma or the extension of a primary lesion of the left uterine adnexa to the bowel. There is no evidence of a benign polypoid tumor of the bowel.

With regard to the differential diagnosis between cancer and diverticulitis, it is at times easy, at times difficult, and occasionally impossible. The age of the patient is not helpful, since either lesion is probable at sixty-five; and the duration of symptoms is quite in keeping with either diagnosis. The absence of clinical symptoms of increasing constipation favors a diagnosis of diverticulitis, whereas the x-ray evidence of complete obstruction leads one to favor cancer, particularly if it persisted after a few weeks of proximal decompression of the bowel. The location of the lesion — the sigmoid area — is a characteristic one for both carcinoma and diverticulitis. The absence of blood in the stools is definitely in favor of diverticulitis, since bleeding is present in only about 16 per cent of the cases of diverticulitis and usually occurs in cases of cancer. The presence of a mass is not helpful. Even at operation one often cannot differentiate the two lesions by inspection or palpation. The pathologist has to decide the matter following resection of the involved area. The x-ray evidence obtained from the first barium enema favors diverticulitis because of the long segment involved (10 cm.) and the absence of destruction of the mucosa. The presence of both lesions is statistically unlikely, as diverticulums have been found associated with carcinoma in only 0.6 per cent of cases, whereas cancer has been found associated with diverticulitis in only 1.8 per cent of cases.

It seems to me that the evidence at hand favors a diagnosis of diverticulitis of the sigmoid with a recent increase in the inflammatory process; an associated localized perforation is a reasonable explanation for the recent x-ray evidence of complete obstruction.

Dr. Tracy B. Mallory: If you had a choice between the two alternatives, Dr. Holmes, which would you choose from the x-ray point of view?

Dr. Holmes: There is no x-ray evidence of tumor. If we made the diagnosis from the x-ray examination alone we should have to say diverticulitis.

Clinical Diagnosis

Diverticulitis.

Dr. Smithwick's Diagnosis

Diverticulitis of sigmoid.

Anatomical Diagnoses

Carcinoma of sigmoid.
Diverticulosis.

Pathological Discussion

Dr. Mallory: This patient was operated on and a segment of the sigmoid was found to contain many large and deep diverticulums. The involved segment was moderately firm. The surgeon's preoperative diagnosis was diverticulitis. When he finished the operation he apparently wavered a bit, since he raised the question of carcinoma; but he still put down diverticulitis as his first diagnosis.

When the sigmoid was opened in the laboratory, a small annular cancer was found in the midst of the segment, which had caused a significant grade of obstruction. We could find no evidence that any of the diverticulums were inflamed, and we thought that they were purely coincidental and that the symptoms were due to carcinoma.

Dr. Smithwick: How do you explain the difference in the barium enema in ten days?

Dr. Mallory: There might have been enough difference in the pressures used during the two examinations.

Dr. Holmes: So one got through and the other did not? I hardly think so. Where was the cancer?

Dr. Mallory: In the midst of the area of diverticulosis in the sigmoid.

Dr. Holmes: At the second examination the obstruction does not seem to be abrupt, and I wonder if they failed to get the enema in.

CASE 29072

Presentation of Case

A thirty-three-year-old man was admitted because of nausea, vomiting, stiff neck and headaches.

Three weeks prior to admission he developed a stiff neck. This was followed ten days later by the onset of severe headaches and then by nausea and vomiting. He soon began to vomit continuously, and was unable to retain anything so that during the week prior to admission he had had no fluids or solids by mouth. Five days before entry he entered a community hospital, where he was found to be afebrile and weak. He became drowsy and lapsed into coma. He was treated with intravenous fluids. A lumbar puncture revealed an initial pressure of 235 mm. of water, and after removing 10 cc. of clear fluid, the pressure was 100 mm.; there were 3 cells per cubic millimeter. Examination of the blood showed a white-cell count of 9200, a red-cell count of 4,000,000 and a hemoglobin of 76 per cent. The coma persisted until the evening prior to admission to this hospital.

The patient was known to have Huntington's chorea, as did his mother and maternal grandmother. For a number of years he had had involuntary twitchings and sudden movements of both arms. He was able to sit quietly, but staggered drunkenly when he walked; he had recently had urinary incontinence. Thirteen years before entry, following a blow to the back of the head, he was said to have suffered concussion. Eight years later he injured his head but was not rendered unconscious. He frequently bumped his head and injured himself.

Physical examination revealed a well-developed, dehydrated semicomatose man lying in bed with his neck hyperextended and stiff, and breathing slowly and deeply through the mouth. The tongue and mucous membranes of the mouth were dry, crusted and caked. The right eye blinked continuously. Both eyes were held with the gaze upward and to the right, although the patient was able to move them voluntarily. The fundi were normal. Examination of the heart was negative. There was dullness with absent breath sounds at the right lung base posteriorly, and a few moist rales were audible at the left base posteriorly. No masses were felt in the abdomen. There was a continuous tremor of the left forearm and the fist was tightly clenched. The tendon reflexes were active. The abdominal and cremasteric reflexes were absent. The Babinski reflex was positive on the right. No clonus could be demonstrated.

The blood pressure was 110 systolic, 80 diastolic. The temperature was 101°F., the pulse 100, and the respirations 25.

The examination of the blood revealed a red-cell count of 5,570,000 with a hemoglobin of 14.6 gm. and a white-cell count of 18,250 with 90 per cent polymorphonuclear leukocytes. The urine was acid in reaction, had a specific gravity of 1.030 and gave a ++ test for albumin; the sediment contained innumerable red and white cells. The urine was unchanged when examined on the succeeding two days. The blood Hinton test was negative. A blood culture revealed no growth. The blood nonprotein nitrogen was 34 mg. per 100 cc., the chloride 115.8 milliequiv. per liter, the total protein 6.8 gm. per 100 cc., the calcium 10.8 mg., and the phosphorus 4.0 mg. A lumbar puncture showed an initial pressure of 110 mm. of water; no cells were found in the spinal fluid, and the gold-sol curve was normal; the protein was 27 mg. per 100 cc. The spinal-fluid Wassermann test was negative. An x-ray film of the chest demonstrated a mottled consolidation in the right lower lung field; there were old scars in the right apex.

The patient was treated with parenteral fluids, and a total of 5 gm. of sodium sulfadiazine was administered intravenously on two days without noteworthy effect. The blood sulfadiazine level on the third hospital day was 4.1 mg. per 100 cc.; the bicarbonate was 25.6 milliequiv. and the chloride 107.7 milliequiv. per liter, and the nonprotein nitrogen was 26 mg. per 100 cc. The patient died on the fourth day after admission.

Differential Diagnosis

Dr. Mandel E. Cohen: To summarize this history briefly, the patient was a young adult who had Huntington's chorea, and it is said that his family had the same trouble. Twenty-one days before admission he had a stiff neck. Ten days before, he had nausea, headache and vomiting and could take nothing by mouth. Five days before, he became drowsy, lapsed into stupor, then coma and apparently when admitted to this hospital was comatose, in fact, we may say he was moribund.

Did the patient have Huntington's chorea, or was that an erroneous diagnosis? All we can do is accept the diagnosis, as it is a well-known syndrome and was present in the patient's family. He was a bit young to have it so far developed, but that is quite all right.

The next question is, Is the incident which led to the patient's death one that is explained by Huntington's chorea, and was it the natural

development of the illness? The answer to that I should say is, No. Patients with uncomplicated Huntington's chorea do not develop sudden acute changes in the central nervous system that cause headache, stiff neck, vomiting and so forth. So I think we shall have to postulate that some other disorder complicating the Huntington's chorea led to the patient's illness and death.

Does the fact that the patient had Huntington's chorea in any way alter the history and the examination? I should say that that certainly is a possibility, because if this man had considerable cerebral change, with psychologic symptoms, it would have been difficult to obtain an accurate history either from him or from his family. Furthermore, the patient probably first consulted his physician late in his illness. There are two other consequences of Huntington's chorea that should be mentioned in connection with this case: one is trauma, and the other is suicide. These patients frequently fall down. A case is recorded in which a man suddenly spun around and fell into a plate-glass window. Another patient, standing near a flight of stairs, had one of the jerky movements of Huntington's chorea, fell over backward and was killed. Suicide is of high incidence in this group, and the question might be raised whether this man had taken some poison, such as bichloride of mercury. We have no evidence for that, but I think we must keep the possibility of trauma in mind, particularly since we are told that he had hurt himself in the past.

I shall assume that some intracranial difficulty led to the patient's final illness and death and that it was a fairly acute process — not something that came as a natural development of Huntington's chorea. There are two good possibilities: one is neoplasm, and the other is subdural hematoma.

In addition we should consider the other types of disease that commonly occur in the brain. First, did the patient have a vascular lesion? Against that are his age and the slow onset. Furthermore, he had no other evidence of vascular disease — no high blood pressure or blood in the spinal fluid. Did he have an infectious lesion in the brain? Here again we do not have evidence for that sort of diagnosis. There was no obvious primary focus of infection. The record states nothing about the ears or sinuses, but I shall assume that they were examined and were normal. He did have a pulmonary lesion, which was detected when he was here but not at the first hospital. Without any further information, I must interpret that as a terminal bronchopneumonia. Against infection are the facts that he was afebrile, that the white-cell count was normal and that the spinal fluid on two occasions showed no cells or elevated protein. I suppose he could, as a remote possibility, have had a brain abscess, but we have no evidence in that direction. Hence, infection was probably not the cause of this man's difficulty.

So that still leaves us with the two possibilities previously mentioned. He probably had a mass of some kind, either a tumor or a subdural hematoma. There is not enough evidence to localize the lesion accurately. Two localizations fit some of the facts, but neither fits all of them. One is that the mass was in the posterior fossa. In favor of that is the fact that the patient had a stiff neck, headache and abnormalities of head posture and eye movements; against it is the fact that the intracranial pressure was not particularly elevated, since the optic disks were not choked. There is some evidence for a more anterior lesion, such as one in the left cerebrum, since the right eye blinked continuously. He had a positive Babinski sign on the right side and tremors on the left — not on the right. One might say that the tremors were due to the chorea and the left-sided lesion had stopped the tremors, but that is just speculation.

I should say that in treating this patient burr holes should be made for a subdural hematoma and that if this were not found a tumor should be looked for. But in making the diagnosis I should place neoplasm first and subdural hematoma second.

If this was a tumor, was it primary or secondary? Metastasis might have occurred from a tumor of the lung. We do not know whether the lesion at the right lower base was due to pneumonia or whether there was some evidence of tumor. In addition, we have not explained satisfactorily the red cells in the urine. Did he have a bladder tumor, which would also account for the incontinence? If we can believe the history, the illness started out of a clear sky with a stiff neck; the red cells were probably due to catheterization on several occasions.

At autopsy, we shall probably be told that in the brain there were the changes characteristic of Huntington's chorea, with atrophy of the cortex and of the caudate nuclei, together with a primary neoplasm, possibly a glioma, which I cannot localize or with a subdural hematoma, and with bronchopneumonia and cystitis.

Dr. Augustus Ross: The importance of a stiff neck in pointing to infection of the central nervous system is worth discussion. Dr. Cohen has assumed that it does not necessarily indicate infection. I should like to ask Dr. Cohen to discuss the mechanism of stiff neck in posterior-fossa tumors.

DR. COHEN: I do not know, and there is no final evidence in the literature. I suppose the general explanation is that such a stiff neck means irritation of the tentorium, which in turn causes the extensor reflexes of the neck to be stimulated; this can occur either with a lesion in the posterior fossa or with a mass in the brain that acts by pressure or herniation.

DR. ROSE: Do you consider this a kind of decerebration?

DR. COHEN: Yes.

CLINICAL DIAGNOSES

Huntington's chorea.
Bronchopneumonia.
Encephalitis?

DR. COHEN'S DIAGNOSES

Huntington's chorea.
Brain tumor (? glioma) or subdural hematoma.
Bronchopneumonia.
Cystitis.

ANATOMICAL DIAGNOSES

(Huntington's chorea.)
Subdural hematomas, bilateral, massive.
Bronchopneumonia, confluent, right lower lobe.
Cystitis, acute hemorrhagic, marked.

PATHOLOGICAL DISCUSSION

DR. CHARLES S. KUBIK: There was a large subdural hematoma on each side, the one on the left being the larger. Since the membranes surrounding the hematomas were fairly dense, the lesions were probably several months old. There was a large temporal pressure cone on the left side, and a smaller one on the right, with resulting compression of the midbrain and aqueduct. There were hemorrhages in the midbrain, a common finding with a temporal pressure cone. Compression of the midbrain and aqueduct probably caused an actual dynamic block, which accounted for the low spinal fluid pressure (110 mm.). Removal of fluid by lumbar puncture would have a tendency to make a bad situation worse by allowing the pressure cone to herniate farther. Pressure against the midbrain or stretching of the tentorium or both may also have had something to do with the stiffness of the neck.

Other changes, presumably related to the Huntington's chorea, were marked atrophy of the caudate nuclei, diminution in the size of the lenticular nuclei and enlargement of the lateral and third ventricles.

Flattening of the cortex by the hematomas made it difficult to estimate the degree of cortical atrophy.

DR. TRACY B. MALLORY: There was a well-marked cystitis, which was certainly the cause of the red cells in the urine and probably the result of catheterization. The lungs showed diffuse congestion and edema, with an area of bronchopneumonia at one base.

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THE VICISSITUDES OF WAR

For the duration of the war the *Journal* has been obliged to adopt changes that are not of its own choosing. Paper is becoming scarce and all publishers of periodicals who buy more than a certain tonnage each quarter have been limited by the War Production Board to 90 per cent of the paper used in 1942. Furthermore, owing to the need for chlorine in the manufacture of munitions and other war materials, the good grades of paper pulp are unobtainable. In addition, the scarcity of manpower is a distinct handicap to the typesetter, the printer, the binder and the mailer. And, finally, the entrance of so many physicians in and around Boston into the armed forces, particularly young men, who are apt to be engaged in clinical or experimental investigative work, has resulted in a marked reduction in the number of contributors to the *Journal*; whereas those who remain at home, for one reason or another, are so deluged with extra duties that the writing of a paper, which to almost everyone is a chore, is necessarily, and probably thankfully, put aside. A reasonable number of papers are still being submitted for publication, but the members of the editorial board are unanimous in their belief that quality should not be sacrificed for quantity — in other words, that the high standards for acceptance should not be lowered.

In the future, the readers of the *Journal* must be content with receiving, somewhat tardily, a periodical of smaller size and one that is printed on a poorer grade of paper. It is anticipated that the two weekly features, "Medical Progress" and the "Case Records of the Massachusetts General Hospital," can be continued, but original articles, like sugar and coffee, must be rationed. For all this, the *Journal* apologizes, but asks that its subscribers accept these unavoidable changes with the same spirit that they have shown regarding the many real sacrifices entailed by the war effort.

PROPHYLACTIC SULFONAMIDE THERAPY

Mechanized warfare and rapidly changing areas of combat often preclude immediate definitive surgical care of war wounds and burns. Various combinations of general and local sulfonamide therapy have been suggested as part of the first-aid routine to delay or prevent the onset of infection during the period of evacuation to competent surgical stations. The British experience at Dunkirk and in the Middle East has established certain facts: wounds filled with sulfanilamide are protected from infection for three to five days only; local sulfanilamide does not prevent staphylococcal wound infection; and wounds excised primarily

heal more kindly than those treated with sulfanilamide and delayed surgical care.[1]

Current experiments are seeking means of prolonging the effect of topically applied sulfonamides and of potentiating sulfonamide action against the staphylococcus. Hawking[2] has used various sulfonamide pastes to prevent bacteriemia in experimental hemolytic streptococcus infection of wounds in rabbits. Sulfanilamide in an oily base was therapeutically superior to watery suspensions of the drug. However, cod-liver oil caused an especially undesirable tissue reaction, liquid paraffin was moderately bad, and even cottonseed oil, the least harmful, had some ill effect. The most successful results were obtained with a suspension in physiologic saline solution of microcrystals of the relatively insoluble sulfathiazole.

Goldberger[3] has reported a potentiated sulfonamide action as a consequence of combination with oxidant antiseptics. Schmelkes and Wyss[4] and Neter[5] have demonstrated that chlorine inactivates the inhibitory effect of para-aminobenzoic acid. This same group of investigators[6] has advanced the hypothesis that the antibacterial effort of sulfonamides is a manifestation of the action of the anionic or zwitterionic species of free dissociated sulfonamide. Inasmuch as all the sulfonamides are weak acids, it has been proposed that a relatively insoluble buffer, such as calcium carbonate, be added to sulfonamide preparations in order to maintain a pH optimal for free dissociation and maximal antibacterial effect. Urea and wetting agents of the triethanolamine type have also been suggested as a means of increasing the solubility and dispersion of topically applied sulfonamides.

Increasing clinical experience with locally applied sulfonamides has recorded instances of excessive absorption of the drug from large wounds and extensive burns. A high blood level of sulfonamide is particularly dangerous when there is depression of renal function from shock or dehydration. Foreign-body reactions and occasional cases of intestinal obstruction have followed the intraperitoneal use of sulfonamides other than the fairly soluble sulfanilamide. The early reports of the treatment of appendiceal peritonitis with subcutaneously and orally administered sulfanilamide have not been significantly bettered as a consequence of the intraperitoneal use of sulfanilamide. There remains the possibility that the success of local applications depends to some extent on the systemic absorption of the drug.

There is every reason to believe that oral or parenteral sulfonamide therapy offers the most effective control of invasive streptococcal infection. Local sulfonamide therapy introduces a factor of uncontrolled drug absorption, and a deleterious effect on wound healing has been reported. Hence it would seem preferable that civilian injuries receive prompt definitive surgical attention and systemic sulfonamide without local drugs. Local chemotherapy should be reserved for those exigencies of battle wherein no other therapy is possible.

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MEDICAL EPONYM

Still's Disease

George Frederick Still (b. 1868), medical registrar and pathologist to the Hospital for Sick Children, Great Ormond Street, London, contributed a paper, entitled "On a Form of Chronic Joint Disease in Children," to the Royal Medical and Chirurgical Society of London on April 23, 1896. The communication was read by Dr. Archibald E. Garrod on November 10, 1896, and appears in the society's *Medico-Chirurgical Transactions* (80: 47-59, 1897).

> There is a disease, occurring in children, and beginning before the second dentition, which is characterised clinically by elastic fusiform enlargement of joints without bony change, and also by enlargement of glands and spleen.
>
> This disease has hitherto been called rheumatoid arthritis, but it differs from that disease in adults, clinically in the absence of bony change, even when

the disease is advanced, and in the enlargement of glands and spleen, and pathologically in the absence, even in an advanced case, of the cartilage changes which are found quite early in that disease, and also in the absence of osteophytic change.

These differences are not to be attributed merely to modification of disease by difference of age, as there occurs also in children a disease in every respect identical with the rheumatoid arthritis of adults.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

EASTON — Elwood T. Easton, M.D., of Boston, died January 31. He was in his sixty-eighth year.

Born in Rockland, he received his degree from Harvard Medical School in 1899. He became ophthalmic surgeon at the Massachusetts Eye and Ear Infirmary, instructor in ophthalmology at Tufts College Medical School and later professor of that department. He was a member of the Massachusetts Medical Society, the American Medical Association and the New England Ophthalmological Society and held the certificate of the American Board of Ophthalmology.

His widow, three sons, three daughters and three grandchildren survive him.

GATES — Ernest A. Gates, M.D., of Springfield, died January 4. He was in his seventy-fourth year.

He was a member of the Massachusetts Medical Society, the American Medical Association and the Springfield Medical Club.

His brother and two sisters survive him.

ROBBINS — Eugene Stanley Robbins, M.D., formerly of New Bedford, died January 18 at West Palm Beach, Florida. He was in his seventy-first year.

Born in Plymouth, Connecticut, Dr. Robbins received his degree from the Bellevue Hospital Medical College, New York, in 1893. He then did postgraduate work in Paris and Berlin. Dr. Robbins was a member of the Massachusetts Medical Society and the American Medical Association.

His widow survives him.

WAR ACTIVITIES

CIVILIAN DEFENSE

Blood-Plasma Reserve

The Medical Division of the United States Office of Civilian Defense pointed out in a recent medical circular that plasma reserves are available in every Civilian Defense region for use in the event of casualties resulting from enemy action or sabotage. The circular emphasizes that this plasma may be used for lifesaving in any disaster. If this plasma is used in nonwar-related incidents, its employment may be considered as a loan, and arrangements may be made later for its replacement, it is pointed out.

Through monthly reports issued by the blood-plasma section of the Medical Division, regional medical officers keep all chiefs of Emergency Medical Service, hospitals and American Red Cross disaster-relief chairmen informed concerning the amount and distribution of plasma reserves available in their states, and how localities may secure additional supplies in emergencies.

In cities where reserves are stored, they may be obtained by hospitals through the local chiefs of Emergency Medical Service. If a community is without plasma or if its supplies are depleted, the local chief may obtain additional plasma in emergencies from the state chief of Emergency Medical Service.

MISCELLANY

HEMORRHAGE FROM THE TRACHEA, BRONCHI AND LUNGS OF NONTUBERCULOUS ORIGIN

"Hemoptysis," wrote French, "literally means blood spitting, but clinically it is restricted to the expectoration of blood from the lungs, bronchi or trachea." Fifty years ago it was pathognomonic of tuberculosis, with mitral disease as the runner up. These two still dominate the field of etiologic possibilities. It has remained for the bronchoscopist (Jackson, C. L., and Diamond, S. Hemorrhage from the trachea, bronchi and lungs of nontuberculous origin. *Am. Rev. Tubere.* 46:126–138, 1942) to show us the prominent roles played by other conditions.

The spitting of blood is, of course, the presenting symptom in many and diverse conditions, so the need for painstaking detailed diagnostic study cannot be stressed too strongly. Short cuts and diagnoses by inference are to be condemned.

First, it is necessary to eliminate hematemesis. Useful here is the fact that blood from the lower respiratory tract is usually frothy and bright red, and apt to be mixed with bronchial secretion, whereas that from the stomach ordinarily is dark and often contains particles of food. It should also be noted that in cases of massive hemorrhage, pallor and loss of consciousness are likely to precede a hematemesis whereas in bronchopulmonary bleeding the blood almost invariably is expectorated before signs of actual blood loss appear.

Having by history and careful physical examination eliminated hematemesis and obvious lesions of the larynx and nasal, oral or pharyngeal cavities, one must assume that the source of the blood is subglottic. It is important to note here that the authors believe that, "far too much emphasis has been placed upon varicose veins at the base of the tongue as hemorrhagic foci." (Not a single case was found in their series.)

Once it is determined that the blood is coming from the lower respiratory tract, tuberculosis is the likeliest diagnosis; to quote the authors, "The disease masquerades under many and varied guises." The inquiry must be considered incomplete until the tuberculous or nontuberculous nature of the underlying lesion has been established beyond question.

Tuberculosis having been ruled out, and cardiovascular disease, the blood dyscrasias and acute lobar pneumonia having been eliminated, the search becomes more difficult.

Precise localization and identification of the causative lesion are dependent on supplementary procedures. A comprehensive fluoroscopic and roentgenographic examination of the chest, including planigraphy and bronchography when indicated, is in order in every case of hemoptysis, as well as bronchoscopy, if necessary. As to the advisability of bronchoscoping a patient during or immediately following a hemorrhage, the authors believe that streaking of the sputum is not a contraindication, but

that when frank hemoptysis has occurred, bronchoscopy should not be performed until several days have elapsed since its cessation.

What now are the etiologic probabilities? The authors indicate them in the accompanying table, which shows the results of careful diagnostic study of 436 patients re-

Tracheal, Bronchial and Pulmonary Lesions Found in 436 Patients with Hemoptysis.

Nature of Lesion	No. of Patients in Each Age Group at Time of Initial Hemoptysis							Total No. of Patients
	Less than 10	10 to 19	20 to 29	30 to 39	40 to 49	50 to 59	60 and over	
Bronchiectasis	19	25	38	19	20	15	2	138
Primary carcinoma of bronchus		1	3	9	20	34	15	82
Tracheobronchitis	4	2	21	12	12	15	8	74
Pulmonary abscess		4	15	16	9	5	2	51
No evidence of disease		2	10	14	5	3		34
Nonsuppurative pneumonitis	2		3	5	3	1	1	15
Suppurative pneumonitis		1	4	1	2	3		11
Adenoma of bronchus		3	3	3	2			11
Secondary cancer of lung				1	1	2	2	6
Lobar atelectasis		2			1	1		4
Primary carcinoma of trachea					1	1		2
Suppurating pneumoconiotic lymph node discharging into bronchus							1	1
Nonspecific granuloma of bronchus				1				1
Streptothricosis						1		1
Chondroma of bronchus						1		1
Osteoma of trachea							1	1
Dermoid cyst communicating with bronchus				1				1
Broncholithiasis							1	1
Neurofibroma involving wall of bronchus		1						1
Totals	25	41	97	82	76	82	33	436

ferred for bronchoscopy. In the interpretation of this table, it is important to note the following comment of the authors:

> A great many patients admitted to the hospital with pulmonary bleeding are not seen by the bronchoscopist, the nature of the underlying disease being such that no indication for the direct inspection of the tracheobronchial tree is present. Included in this category are patients with cardiovascular lesions which lead to the production of chronic passive congestion or pulmonary infarction, patients with acute lobar pneumonia, and patients with blood dyscrasias. This group, observed by the internist alone, represents a considerable number of patients with hemoptysis.

The authors make the following conclusions:

> Inflammatory processes are responsible for the hemorrhage in the majority of the cases, the most common etiologic agent being bronchiectasis. When one takes into consideration the fact that expectoration of blood is the initial manifestation of carcinoma of the bronchus in only a very small percentage of the patients, it is obvious that bronchoscopy must be done and the diagnosis made early in the course of the disease, before the symptoms have reached the stage of hemorrhage, if a successful therapeutic result is to be achieved in these cases.
>
> Fatal hemorrhage occurred in but 3 of the patients in the series, each of whom had a pulmonary abscess. —Reprinted, in part, from *Tuberculosis Abstracts* (February, 1943).

ANNUAL PRIZE SUBSCRIPTION

The annual prize subscription offered by the *New England Journal of Medicine* for the best undergraduate contribution to the *Tufts Medical Journal* has been awarded to E. Allen Joslyn, '44, for his paper "Fat Metabolism in Relation to Diabetes," which appeared in the December, 1942, issue. The paper, "Cardiac Surgery," by Mauray Tye, '43, received honorable mention; it appeared in the October, 1942, issue of the journal.

BOOK REVIEWS

Surgery of the Ambulatory Patient. By L. Kraeer Ferguson, M.D. With a section on fractures by Louis Kaplan, M.D. 8°, cloth, 923 pp., with 645 illustrations. Philadelphia: J. B. Lippincott, 1942. $10.00.

The purpose of this book is clearly announced in the challenging statement of the authors, "It is probable that more poor surgery is performed on minor lesions of the hand than, for instance, on major brain lesions." For the surgeon who has mastered the technic of major surgery, much of the general discussion is superfluous. But this book will be of greatest help to the general practitioner who only does surgery on minor cases and in whose hands a minor operation may become a major disaster if certain precautions are not followed.

Although many of the procedures described may be done on office or hospital outpatients, some are better reserved for hospital inpatients. The author states that he has ligated over two hundred saphenous veins as an office procedure. Some surgeons are emphatic in their insistence that this should always be done in the hospital as a full-dress operation. However, this is in no way a criticism of the basic material in the book, which is eminently sound. The division into symptoms, pathology, diagnosis and treatment makes each section easy to follow, and the illustrations are so well chosen and in such numbers that one can often follow the author by means of them, without reference to the text.

In a work such as this, one hesitates to single out any one portion as superior to the others. However, the considerations of breast pathology, low-back pain and varicose veins are so good that they seem to deserve special mention.

Ambassadors in White: The story of American tropical medicine. By Charles M. Wilson. 8°, cloth, 372 pp., with 42 illustrations. New York: Henry Holt and Company, 1942. $3.50.

The story of American tropical medicine is told by Wilson in an exciting and enthusiastic style. Most of the story will be news, not only to the layman but also to the physician who does not know the tropics. The names of Gorgas, Walter Reed and Noguchi are familiar to all, but how many of us have more than an inkling of what they did? Still fewer know about the pioneering of Finlay in yellow fever or have heard of the great medical service built up by Deeks for the United Fruit Company.

In his first chapter, Wilson emphasizes the transcendent significance of disease in tropical America. Although what the author says is true of the peons in certain primitive and unsanitated regions, it is not true of the great cities of Latin America or of the more cultured elements of the population. In other chapters, the health problems

arising from a few of the more important diseases of the tropics are presented briefly. There are also vivid sketches of outstanding personalities upon whom praise has been generously bestowed. A few inaccuracies of no great importance were noted by the reviewer, and some of the information about disease is out of date.

The reviewer regrets being obliged to point out that on pages 200, 201 and 202, in his sketch of Noguchi's work, Wilson has criticized unfairly the research of a Harvard expedition to Peru. Not only has the author been ill-informed, but the tone of some of his remarks is offensive. Noguchi would never have assumed this attitude. Noguchi, moreover, would have recognized the fact that, at a time when his *Leptospira icteroides* had received general acceptance as the cause of yellow fever, skepticism persisted at Harvard. This skepticism culminated in a series of well-planned experiments by Sellards and Theiler which proved beyond a shadow of doubt in 1926 and 1927 that *Leptospira icteroides* and *L. icterohaemorrhagiae* were identical, and that a leptospira was not the cause of yellow fever.

Theiler, M., and Sellards, A. W. The relationship of *L. icterohaemorrhagiae* and *L. icteroides* as determined by the Pfeiffer phenomenon in guinea pigs. *Am. J. Trop. Med.* 6:383-402, 1926.
Sellards, A. W. The Pfeiffer reaction with leptospira in yellow fever. *Am. J. Trop. Med.* 7:71-95, 1927.
Sellards, A. W., and Gay, D. M. The fate of *Leptospira icteroides* and *Leptospira icterohaemorrhagiae* in the mosquito, *Aëdes aegypti*. *Ann. Trop. Med.* 21:321-342, 1927.
Sellards, A. W., and Theiler, M. Pfeiffer reaction and protection tests in leptospiral jaundice (Weil's disease) with *Leptospira icterohaemorrhagiae* and *Leptospira icteroides*. *Am. J. Trop. Med.* 7:369-381, 1927.
Sellards, A. W. The relation between Weil's disease and yellow fever. *Ann. Trop. Med.* 21:245-259, 1927.

Acute Injuries of the Head: Their diagnosis, treatment, complications and sequels. By G. F. Rowbotham, B.Sc. (Manch.), F.R.C.S. (Eng.). With a foreword by Norman M. Dott, M.B., Ch.B. (Ed.), F.R.C.S. (Ed.). 8°, cloth, 288 pp., with 124 illustrations. Baltimore: The Williams and Wilkins Company, 1942. $7.50.

"This book is written primarily for those who are responsible for the treatment of acute cerebral trauma and who have not received special training in neurosurgery or neurology." Every hospital must care for patients with acute craniocerebral injuries, and usually the small institutions do not afford good treatment because of a disinterested profession. It is essential that general surgeons who are called on to treat such patients know the main points of diagnosis on which treatment is based. They should recognize the indications for exploration of the cranial vault, for subtemporal decompression and for the treatment of compound fractures of the skull. This book furnishes a concise review, which may serve as a guide to the correct handling of craniocerebral injuries.

There are some well-chosen illustrations on operative technic. The last chapter, "Sequels of Head Injuries," is the best of the whole book. This aspect is one of the most important but often most neglected of the entire subject. The description of the post-traumatic syndrome could not be better done that it is in the author's depiction of such a patient in the physician's office.

A few items in the book deserve mention because of their variance with American practice. The author does not encourage, but he does allow, the use of morphine after head injuries in ¼-gr. doses; whereas in this country the drug is considered to be absolutely contraindicated in such cases. In the author's experience of 25 cases of middle meningeal hemorrhage, all had a lucid interval; the many exceptions to this are not stressed sufficiently. It is stated that patients with craniocerebral injuries can usually be up and about in three weeks, with gradual mobilization during this period. In the reviewer's experience, probably no single factor can prevent development of the postconcussion syndrome more than absolute rest in bed; in severe injuries this should be continued for at least three weeks, and as long after that as symptoms are present.

Handbook of Health for Overseas Service. By George C. Shattuck, M.D., with contributions by other authors. 24°, cloth, 198 pp., with 14 illustrations. Cambridge, Massachusetts: Harvard University Press, 1942. Not for sale (copies may be obtained gratis from the Harvard University Press).

This volume was prepared for the Co-ordinator of Information, Washington, D. C., now known as the Office of Strategic Services. Its purpose is to act as a guide for nonmedical men who travel in places where medical services are not available. It covers such subjects as food, drinking water, camp hygiene, clothing and first aid, with a brief discussion of many diseased conditions that may be encountered. Although all diseases are not included, it is reasonably comprehensive, covering ailments of the tropical and arctic regions. A list of the drugs and surgical equipment that should be available are included, although these necessarily are limited in number.

The volume is compact, has a flexible cover and can easily be carried in the pocket. It should be of great value to men going on field service and can safely be recommended by the physician to patients so situated that immediate medical attention is not available.

Doctor Bard of Hyde Park: The famous physician of Revolutionary times, the man who saved Washington's life. By John B. Langstaff. Introduction by Nicholas M. Butler. 8°, cloth, 365 pp., with 11 illustrations and frontispiece. New York: E. P. Dutton and Company, Incorporated, 1942. $3.75.

This is a carefully written and well-documented life of one of the outstanding Revolutionary physicians who practiced in New York City during the latter part of the eighteenth century. He was a physician at one time to George Washington and served as president of the College of Physicians and Surgeons. He later retired to Hyde Park on the Hudson, and the title of the book is taken from this relatively unimportant period of his life. The interest in Bard is largely through the development of the New York Hospital, his operation on George Washington for a severe carbuncle and the pleasant stamp that he gave to medicine during his time. He was an excellent example of the English gentleman physician, in direct line with Lettsom and Fothergill. The author has done an excellent piece of work in collecting the data. He has long been associated with the medical profession for he has served as chaplain of the Bellevue Hospital and is the son of a well-known physician. He descends, moreover, from physicians who were active in New York in Bard's time. This is an important biography and should be widely read.

(Notices on page xiii)